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JANUARY-DECEMBER, 1920

With [illegible]3 Plates, and 112 Text Figures

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WILLIAM GILSON FARLOW

PHYTOPATHOLOGY

VOLUME X NUMBER 1

JANUARY, 1920

# WILLIAM GILSON FARLOW

G. P. CLINTON

WITH PORTRAIT, PLATE I

Professor William Gilson Farlow was born in the vicinity of Boston, Mass., on December 17, 1844. He died in Cambridge, Mass., on June 3, 1919, in his seventy-fifth year. His death occurred after a comparatively short illness, at his residence of recent years on Quincy Street opposite the main yard of Harvard University, his beloved Alma Mater. Although retired from active teaching for many years, Professor Farlow continued to the last in his work at the Cryptogamic Herbarium which he founded, supported financially during his life, and to which on his death he gave his library, botanical specimens, and an endowment fund.

Many things contributed to make Professor Farlow very successful in his life work—family, wealth, training, social prestige, position, native ability, and systematic and continued effort, all had their influence. He greatly loved his work, otherwise he would have taken things easier during his declining years. He was very fortunate in the teachers under whom he studied, for in their time there were none at least to exceed Gray and deBary. His ability was evident to anyone who came in personal contact with him. To the mind of the writer these three factors, love of his work, sound training, and native ability, were chiefly responsible in making him the foremost cryptogamic botanist of America. Furthermore, at the time of his death he was generally considered by his fellow botanists as Dean of the active American botanists, as indicated by length of service, honors, etc.

Professor Farlow graduated from Harvard College in 1866 and from the medical department of the University in 1870. His interest in botany induced Doctor Gray to make him his assistant in cryptogamic botany. After two years of teaching, young Farlow decided that he needed more training in this subject, and such not being available in America, as he was a pioneer teacher here himself, he went to Europe, studying in Germany and France. While under deBary at Strassburg he came in contact not only with this great teacher and investigator, but also with a number of his students from different parts of Europe, who, in after years, became leaders in various lines of botanical work. This experience no doubt greatly influenced his own later development.

Stimulated by his European training of two years, he came back to Harvard and was made Assistant Professor of Botany, and stationed at the Bussey Institution at Jamaica Plains. It was here that he began a series of articles on plant diseases that eventually included investigations of the potato rot, black knot, downy mildew of grape, smut of onion and the relationships of Roestelia and Gymnosporangium, the first paper appearing in 1875. No matter how closely Harvard cryptogamic botanists in later years have clung to the pure side of this science, one cannot get around the fact that at the start the applied side was ably handled by Farlow, and that his earliest students also published similar articles. As this was at a time when our state institutions, with only one or two exceptions, had not manifested any particular interest along this line, we can justly claim that Farlow, with Burrill, was really the pioneer plant pathologist of America, although he may be antedated by others as mycologists.

Not only was Farlow doing investigational work at this time, but he was actively engaged in teaching at Bussey and also at Cambridge in both the regular and summer school courses. His first advanced student was Byron D. Halsted, a recent graduate of Michigan Agricultural College, and the first to obtain a doctor's degree under him. Since then many American botanists have had the privilege of receiving part of their botanical training under his immediate direction or in the department he created and still served in an advisory way till his death.

In 1879, the funds of Bussey becoming diminished, Farlow was transferred to Harvard University proper and made Professor of Cryptogamic Botany. Here he continued under the varying changes of the University and the times, building up the teaching department, creating the Cryptogamic Herbarium, training scientific men who might give their undivided time to the subject, either as teachers or investigators or as both, publishing botanical articles of merit, and advancing the interests of cryptogamic botany throughout the country as has no other man. Remembering that he started in at a time when there were few men in the country interested in this branch of botany, that there was no teacher who gave it much attention in his classes, that laboratory work or equipment for such was not to be had, and that he lived to see the day when it really predominates the science of botany in the United States and when many men make it their life work and get their living out of it (I say "living" not necessarily meaning anything more), is it any wonder that he was interested in his work or that botanists generally honor him for the great part he had in its development?

And honors did come to Farlow from his own and other universities of this country and Europe, not only through honorary degrees but in other ways as well. The various prominent scientific societies of the world had him enrolled as a member and he became president, at one time or another, of most of those in America. Of honors obtained we need mention only a few—Member of the National Academy of Science, London Linnaean Society, British Association for the Advancement of Science; President of the A. A. A. S., Botanical Society of America, American Naturalists; honorary degrees from Harvard University, University of Wisconsin, University of Glasgow, University of Upsala.

In middle life Professor Farlow was married to Lillian Horsford, who still survives him. To visiting botanists whom Farlow aided in their examination of specimens at the Herbarium, there was usually added the pleasure of entertainment at his home and an inspection of his botanical library which was kept there. In this way Mrs. Farlow came to know many of his fellow-workers, and the charm of her personality was appreciated by all.

We have so far recorded in a general way a few of the main facts in the life of Doctor Farlow. The time given for the preparation of this article has been too short to allow us to present facts or incidents that are not already at hand. Neither do we wish to make this paper too much in the nature of a formal biography but rather as the loving remembrance of one who, through twenty-five years of correspondence and two years of student life, feels that he owes a debt of gratitude, for favors received and inspirations aroused, to a master of high order. It seems fitting, therefore, that we write further concerning Doctor Farlow as a man and as a scientist, and of his service to the science of botany.

If in speaking of him as a man the writer touches on intimate things no offense is intended, and his excuse is that we usually respect men as much for what they are in their natural selves as for the names they have attained. Farlow was quite short in stature, though well proportioned. When we first met him his hair was turning grey, but his cropped mustache and shaggy eye-brows, with a considerable tinge of red, suggested that in his youth he might have been classed as red-headed. He had a decided roll, or side glance, of his eyes in talking, and what might possibly be termed a Boston or English inflection in his speech, which was rather rapid, so that one had to keep his ears and mind alert to catch all he said. The writer can see him now as he looked each morning after his arrival at the Herbarium making his daily pilgrimage to the shrine of Thaxter, passing rapidly through the advanced laboratory, taking in at a glance the students there, perhaps recognizing some one with a nod, and disappearing into the chamber of consecrated and concentrated work, thence to emerge shortly afterward with Thaxter escorting him back to his room—the short and the tall—earnestly engaged in some discussion, each mutually dependent on and helpful to the other! Such earnest comradeship devoid of the ordinary frivolities of men was charming to behold.

We must admit that dignity was quite an attribute of Farlow. Those who have seen him writing his letters, as he sometimes did, standing at his desk, did not take this to be unusual for it caused him no inconvenience, such as most of us would have had. That he knew how to put people in their proper places is illustrated by the remark that a "character," who was claiming newspaper fame through his discovery of fungous fairy rings, let out in the laboratory one day: "He may be small but one may not presume to impose on him on that account, except to his sorrow." His dignified attitude is further shown by the following story he told on himself: It seems that a western botanist was calling on him at an inconvenient time and during very warm weather. On their way to the Gray Herbarium, to which Farlow was taking him, this botanist as they passed through Cambridge Common took off his coat and lay down on the grass, and suggested that Farlow follow suit. It was the informality of the west meeting the formality of the east, and neither could understand the other. For, as Farlow later said, "What would people think of me if they saw me doing this." When one is in Cambridge one must do as the Cambridge professors do, is the answer.

Still, with all his reserve, Farlow was witty and saw the humorous side of things. He was apt to use this wit in a refined and semi-hidden manner by way of suggestion to a person with whom he might be talking, so that one was not always sure just when he came in for his share of mild criticism. Among the Harvard professors, the writer was once told, Farlow shared honors with another member of the faculty as being the wittiest man there. Those who heard him give, or have read, any of his general addresses, will appreciate this fact. Especially interesting to all present was the presidential address he gave at the botanists' dinner at Cleveland in 1912.

He was somewhat critical in his judgment of men and their work. Perhaps this was partially responsible for the feeling that has gone out that Harvard does not wish great numbers of advanced students so much as a few of unusual promise. He did not judge others or their work, however, with undue harshness but rather his extended experience in reviewing literature—for he went over, at least in a general way, all of the current botanical articles—gave him the ability to pick out the weak points with little effort. In discussing this or that botanist with him, if one did not know this, he might misjudge Farlow's real estimate of the man under discussion.

His letters also sometimes show this critical attitude, in fact, even when writing of one of whom he thought highly. The following extract from an old letter (the italics being used for camouflage) illustrates the point under discussion: "*Smith* was in Cambridge for a few days. I am sorry that he did not make a very favorable impression. He looked over the covers of *two genera of fungi* but made no microscopic examination and seemed content to make a copy of hosts and localities without making any critical study of the specimens. He did not look at any of the exsiccati and seemed displeased that he could not have all the specimens of the two genera laid out before him at once. Evidently he had no idea of what was in large sets of exsiccati and I could not, to save my life, make out what he wanted and could only infer that he did not know what he wanted himself. He made me a present of about half a dozen specimens. I wonder whether he thought I did not have plenty of our common species," etc. And this was a botanist who had made a name of some prominence, but you cannot guess who he was from this tale.

Professor Farlow liked to hear of the current gossip about botanists and botanical things, and if this did not come through ordinary conversational channels, he occasionally went after it by direct inquiry. In this way he kept in touch with men whom he had not met or seen for some time, and with events with which he had no very direct connection. Chestnut Blight and White Pine Blister Rust investigations were two things that thus solicited his interest, and the writer would like to print some of his spicy comments just to show how interested he became in subjects that mix politics, advertising and investigational work more or less together.

We have dwelt thus at length on this personal side of his life to show that he was a human as well as a dignified Harvard professor, and that he had a personality that one always likes to find in a great man, to give him color as it were. So whenever his former students got together he was always brought up for discussion, and his ears, figuratively speaking, must have often tingled from the personal comments that followed, showing in what high regard he was held by all.

As a scientist we must judge him by the articles he published, and here the writer ventures a criticism of a man who was himself a critic. He published *too little* for what ke knew and for what he had done. This is not a criticism that can be made of many botanists, however. It was voiced in his presence once by a professor who did not mean at all to be sarcastic, when he said: "In one of your recent publications, Doctor Farlow, published some ten years ago," etc. The writer believes that every American botanist regrets that Doctor Farlow did not leave on record in published papers more of his vast knowledge of lichen[illegible]sses, algae, and fungi, knowledge that was so extensive and exact t[illegible]n authority on any of these groups was bound to pay profound re[illegible]ce to his opinions concerning the same. We have, or have had, bota[illegible]ist who surpassed him in knowledge concerning each of these groups, sav[illegible] fungi, but none who equalled him in familiarity with them all. H[illegible]ws truly entitled Professor of Cryptogamic Botany. Just as surely as [illegible]old time Professor of Natural History has passed out of existence, s[illegible] the mind of the writer, he represents the last of those who were able s[illegible]ctorily to cover the wide field of cryptogamic botany.

Aside from his New England Algae and a few similar [illegible], he published little concerning the chlorophyll-bearing cryptogar[illegible] However, if he did not publish along these lines, he helped and stim[illegible] and others to do so. His papers on the fungi were more numerous a[illegible]d of high grade. They were true contributions to our knowledge of the [illegible] whether made in the form of short notes on personal collections or [illegible] extended morphological and systematic articles. They covered a [illegible] range of subjects for he considered all genera as worth knowing. [illegible] writer has always thought, however, that he was more interested [illegible] rusts than in other groups.

No doubt his greatest contributions, at least in si[illegible]d general value, were those concerning the bibliography of fungi. St[illegible] in 1887 he issued, with Trelease, a very complete bibliography of [illegible]ticles on American fungi published before that year, and this was fo[illegible] the next year by a supplementary list, by Farlow alone, of addend[illegible] those articles published in 1887. Critical notes add greatly to the [illegible] of this work. In 1888, with Seymour, he published the Host Ind[illegible] Fungi in the United States. This work has been of even greater [illegible] to botanists, especially as an aid to identification of species. It s[illegible] be brought up to date by Thaxter and Seymour as a memorial vol[illegible] Farlow. His most pretentious work, however, is the Bibliograph[illegible]dex of North American Fungi, the first part of the first volume bein[illegible]ed through the Carnegie Institution in 1905. The immensity of th[illegible]k is shown by the fact that the genera treated, arranged alphabet[illegible], include only those from Abrothallus to Badhamia. It is the earn[illegible]pe of all botanists that this valuable work may be continued and [illegible] provision has been made for the early appearance of subsequent pa[illegible]

Finally, what was Farlow's influence on the science [illegible]ny in general, and cryptogamic botany in particular in the Unite[illegible]s? This has already been indicated in a general way. We wish to [illegible]te it in a little more precise form under the following headings:

1. He was a pioneer investigator in plant pathology and so helped this branch to obtain needed recognition in the botanical world at a time when botanists were given too largely to the consideration of the morphology and systematic classification of flowering plants. In his investigations along the lines of pure science he set a high standard for accuracy of statement and originality of information. In his bibliographical compilations of the literature of fungi he has given aid of great value to all other investigators.

2. In the Harvard Cryptogamic Laboratories and Herbarium he started an institution new of its kind in this country and for many years leading in facilities for under-graduate and post-graduate work all similar institutions that followed. The high ideals of fundamental and thorough instruction there put in practice have been ably carried out by his successors and assistants.

3. He turned out as students from these laboratories many of the leading American botanists. Through his wonderful library and the herbarium he aided visiting botanists from time to time to obtain information and see specimens not generally available. He brought together in the herbarium one of the most complete collections of exsiccati of fungi that exists today in the world. Through his own collections, specimens sent him, and the purchase or donation of such valuable collections as those of Curtis' fungi, Tuckerman's lichens, Sullivant's and James' hepatics and mosses, and algae by many noted collectors, he has made this herbarium a mecca for botanists interested in many lines of work.

4. Through his attendance at botanical conferences in this country and Europe, and through published opinions on the question of botanical nomenclature, he has exerted a strong influence in the form of a sane and stable use of scientific names as applied to fungi. Through this conservative treatment, as compared with the more radical views held by some in this country, he has maintained a connecting link with European botanical views on this subject, which we hope some day may lead to uniformity of usage everywhere.

5. Finally, through correspondence with botanists all over the country, aiding them by looking up references in unusual publications, sending specimens from his herbarium for their benefit, or determining specimens sent him, he has given great aid in furthering the investigations of others. This is a sort of work that often goes unappreciated by botanists, and perhaps explains in a measure why he did not publish more himself, as he gave much of his time to others. We have before us a hundred or more letters, all in his hand writing, many of them giving information asked for and requiring effort and care on his part to obtain. If to these are added the hundreds of letters he has written to others in the same helpful way, we gain a truer insight into his influence on botanists and their work.

The writer had the privilege last March, shortly before Doctor Farlow's final illness, of spending part of the day at the herbarium and in his library obtaining desired information covering certain smuts. As always, there was the same chatty talk concerning botanists, a little warning not to stir up a mutual friend too much about the war, the furnishing of desired specimens, and the repeated bringing of reference books opened at the designated page. It is a pleasure thus to think of him still at work in his beloved herbarium and library, doing favors for others.

CONNECTICUT AGRICULTURAL EXPERIMENT STATION,
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# STUDIES WITH MACROSPORIUM FROM TOMATOES

J. Rosenbaum

With Plates II and III and One Figure in the Text

The early blight or "nail head" spot (*Macrosporium sp.*) on tomato fruit has been known to growers and investigated by pathologists for a number of years. It is still a question, however, whether:

(1) Infection in the field can take place without previous injury to the fruit,

(2) The disease can originate and spread in transit on winter-grown tomatoes in the South,

(3) The same or different species of Macrosporium cause similar or different lesions in the various tomato growing sections,

(4) Macrosporium on tomatoes is identical with *M. solani* as found on the potato.

The work here reported was done during the past three years with winter-grown tomatoes in southern Florida. Additional observations were made near Washington and in some New Jersey fields. Diseased specimens for study and isolations were also collected or received from Texas, Louisiana, Tennessee, and Maryland.

The great destructiveness of this disease in the field has been recorded by Edgerton and Moreland (1), Sherbakoff (2), and more recently by Hesler (3).

Edgerton and Moreland (1) say: "It probably stands as a close second to the wilt in destructiveness, and in many regions it is the all important disease. . . . . At Norwood, where there were over 200 acres in tomatoes, the loss due to this disease was estimated to be at least 50 per cent."

Sherbakoff (2) states: "An accurate estimate of the damage caused by it is not available, but it is safe to say that wherever the disease is present a loss up to one-half the crop is sustained, even during a comparatively dry season. A much heavier loss is not rare." Hesler (3) states: "Growers and others agree that a least 20 per cent of the fruits are culled out at the packing house on account of blight lesions. In addition to this loss, many tomatoes are not picked on account of early blight spots." The observations made by the writer lead him to conclude that in southern Florida where, according to the Crop Estimates Report (9) 12,925 acres are grown, this disease is by far the most destructive and widespread with which the growers have to contend. In a letter received from one of the growers, referring to this disease the following statement is made: "I have lost in the past eight years 20,000 crates."

*Differences between old and new spots.* The difference in appearance between an old spot and one resulting from a relatively new infection has enabled the writer to make numerous counts in the field with a view to determining whether the size and age of a fruit have any relation to infection.

The young spots are superficial, light brown in color, with an indistinct margin. With age the lesions become more and more distinct, the color gradually changing from light brown to dark brown or gray, with the margin becoming more definite. The majority of the spots after they have attained the size of a nail head have reached the maximum size. They remain in this condition unless other fungi gain an entrance. With age the spot shows a break or crack. It is this opening which affords entrance for other fungi and causes the lesion to enlarge.

There is a period of four to eight weeks from the time the spot is first visible to the time when it takes on the appearance of an old spot, that is, becomes dark brown in color, sunken, and possibly shows a distinct break in the skin.

*Time of infection in the field.* With the above knowledge as to the differences in appearance of an old and new spot, counts were made from a number of badly infected fields. In all cases the circumference[1] of the fruit was measured and the appearance of new spots noted. After making some preliminary measurements and counts it was decided to divide the fruits into those six inches or less in circumference and those more than six inches in circumference.

In table 1 are given the counts made from a badly infected field, part of which was sprayed with Bordeaux after the first picking of fruit had been made. All the fruit from the size of a marble to mature fruit was picked. It shows therefore, not only the new infections on the small and large fruits, but incidentally also the effect of spraying on the severity of the infection, the percentage of disease in the sprayed part being 51.4 as compared to 79.6 per cent in the unsprayed. The increase in the quantity of diseased fruit from December 22, when the first count was made, to January 5, when the second count was made, was almost 54 per cent. On December 22, 1917, 174 tomatoes were picked from another field where the

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[1] Circumference of the fruit referred to throughout the paper refers to the polar circumference, i.e., in taking the measurement the tape passed around the stem and blossom end of the fruit.

TABLE 1

*Development of Macrosporium on large and small fruit and effect of one spraying*

| DATE PICKED | NUMBER OF PLANTS | TREATMENT | NUMBER OF CLEAN FRUIT |  | NUMBER OF FRUIT WITH OLD SPOTS |  | NUMBER OF FRUIT WITH NEW SPOTS |  | PER CENT DISEASE NOT SPRAYED | PER CENT DISEASE SPRAYED |
|---|---|---|---|---|---|---|---|---|---|---|
|  |  |  | Circumference more than 6 inches | Circumference less than 6 inches | Circumference more than 6 inches | Circumference less than 6 inches | Circumference more than 6 inches | Circumference less than 6 inches |  |  |
| December 22 ..... | 30 | Unsprayed | 107 | 76 | 0 | 0 | 6 | 58 | 25.9 |  |
| January 5 ........ | 30 | Unsprayed | 39 | 12 | 20 | 0 | 0 | 179 | 79.6 |  |
| January 5. ....... | 15 | Sprayed | 40 | 80 | 14 | 0 | 0 | 113 |  | 51.4 |
| Total........... | 75 |  | 186 | 168 | 34 | 0 | 6 | 350 |  | 52.4 |

disease was present. All the fruit from the size of a marble and up was picked and immediately sorted as follows:

|  |  |
|---|---|
| More than 6 inches in circumference, healthy | 67 |
| More than 6 inches in circumference, showing new infection | 0 |
| Less than 6 inches in circumference, healthy | 54 |
| Less than 6 inches in circumference, showing new infection | 42 |
| Less than 6 inches in circumference, showing older infection | 11 |
| Per cent of new infection in large fruit | 0 |
| Per cent of new infection in small fruit | 39.2 |

To determine whether new infections would show up on the fruit after being held, an equal number of clean, small and large fruits was picked December 22, and kept at the laboratory for seven days. At the end of that time counts were made with the following results:

|  |  |
|---|---|
| Fruits more than 6 inches in circumference remained healthy | 77 |
| The fruit less than 6 inches in circumference showed: |  |
| Fruits with one new infection | 13 |
| Fruits with two new infections | 7 |
| Fruits with numerous new infections | 41 |
| Fruits remaining healthy | 16 |
| Per cent of fruit more than 6 inches in circumference developing new spots | 0 |
| Per cent of fruit less than 6 inches in circumference developing new spots | 79.2 |

A repetition of the above counts was made April 12. In this case three baskets of immature fruit varying from 1 to 6 inches in circumference were picked and carefully examined for early blight spots. Those showing any suspicious or definite lesions had these marked by ringing with india ink and were in this way excluded from the final counts. The fruit was examined April 17 and April 22 for the development of new spots. The results are shown in table 2.

Seventy-five fruits more than 6 inches in circumference picked at the same time and kept under identical conditions remained healthy.

For comparison the salient points in the above counts have been brought together and summarized in table 3. The figures show conclusively that new infections in the field very seldom occur on mature fruit or fruit more than 6 inches in circumference.

TABLE 2

*Development of Macrosporium on immature fruit*

| NUMBER OF NEW SPOTS DEVELOPED | NUMBER OF FRUIT IN BASKET 1 |  | NUMBER OF FRUIT IN BASKET 2 |  | NUMBER OF FRUIT IN BASKET 3 |  | TOTAL FRUIT |  |
|---|---|---|---|---|---|---|---|---|
|  | April 17 | April 22 | April 17 | April 22 | April 17 | April 22 | April 17 | April 22 |
| 1 spot | 10 | 12 | 4 | 5 | 19 | 9 | 33 | 26 |
| 2 spots | 13 | 9 | 12 | 4 | 9 | 9 | 31 | 22 |
| 3 spots | 8 | 5 | 3 | 3 | 4 | 8 | 15 | 16 |
| More than 3 spots | 31 | 15 | 40 | 60 | 38 | 19 | 118 | 51 |
| Remaining clean | 9 | 3 | 11 | 4 | 9 | 4 | 29 | 11 |

TABLE 3

*Development of Macrosporium on mature and immature fruit*

| COUNT NUMBER | MATURE FRUIT |  |  | IMMATURE FRUIT |  |  |
|---|---|---|---|---|---|---|
|  | Number healthy | Number showing new infection | Per cent diseased | Number healthy | Number showing new infection | Per cent diseased |
| I | 186 | 6 | 3.1 | 168 | 350 | 67.5 |
| II | 67 | 0 | 0 | 51 | 12 | 39.2 |
| III | 77 | 0 | 0 | 16 | 61 | 79.2 |
| IV | 68 | 0 | 0 | 11 | 218 | 95.1 |

*Shipment of fruit.*[2] Some growers have been of the opinion that the early blight spot can originate in transit. In every case, however, where a shipment of infected fruit was traced to the point of origin it was found that the fruit was spotted at the time of packing. Rands (1) found the disease in the Chicago and Madison markets on tomato fruit from the South

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[2] Assistance in the examination of the fruit at the point of arrival was given by Mr. G. H. Godfrey, Dr. N. E. Stevens, Dr. V. B. Stewart, Dr. Geo. K. K. Link, Mr. Pomeroy and Mr. Van Orman.

He says, "The evidence here indicated that the disease had developed during transit."

Throughout the winter season, December to May, 1917, crate shipments of tomatoes were made to Washington and New York City. Nail head spots on fruit from badly infected fields as well as from clean fields, were marked by ringing with India ink before packing.

A total of 17 express and freight shipments were made, involving 75 crates. The results of these shipments showed that mature fruit picked green, as is usually done, and healthy at the time of packing, showed no new spots on their arrival. Moreover, fruit badly spotted at the time of packing and packed in such a way that diseased and healthy fruit alternated showed no spread of the disease. This was done both with tomatoes wrapped and unwrapped.

TABLE 4

*Development of Macrosporium on mature and immature fruit in transit*

| NUMBER OF NEW SPOTS | IMMATURED FRUIT | | | MATURED FRUIT | | | PER CENT DISEASED | |
|---|---|---|---|---|---|---|---|---|
| | Basket 1 | Basket 2 | Total | Basket 3 | Basket 4 | Total | Immature | Mature |
| 0 | 14 | 4 | 18 | 24 | 23 | 47 | | |
| 1 | 14 | 8 | 22 | 0 | 1 | 1 | | |
| 2 | 13 | 6 | 19 | 0 | 0 | 0 | | |
| 3 | 19 | 5 | 24 | 0 | 0 | 0 | | |
| 4 | 5 | 3 | 8 | 0 | 0 | 0 | | |
| 5 or more | 26 | 33 | 59 | 0 | 0 | 0 | | |
| Total . | 77 | 55 | 132 | 0 | 1 | 1 | 88 | 2 |

On the other hand, all shipments of immature fruit, that is, fruit less than 6 inches in circumference, picked green and clean from an infected field, developed numerous spots at the time or soon after their arrival.

In table 4 the counts are given from one of the shipments. The shipment was made April 14 from Miami, Florida, in a car containing over 400 crates of tomatoes, and was examined at New York City ten days later. Repetition of this work gave similar results and confirmed the evidence of counts made in the field, that mature fruit as indicated by the size never becomes infected.

*Artificial infections.* The very excessive spotting of fruit in badly infected fields and careful examination of such fruit would seem to indicate that natural infection in the field takes place without previous injury to the tomato. Nevertheless, the writer heretofore had not been able to produce infection without injury to the fruit. This has been the experience of previous investigators as shown by the statements of Rands (4), McCubbin (5) and Jones and Grout (6), all of whom were unable to produce typical symptoms on the fruit without previous injury. In view of the observations made in the field that infection takes place on the very young fruit, the idea suggested itself that artificial infection without previous injury could be obtained by inoculating young or immature fruit.

Clean fruits of various sizes were collected and placed in moist chambers and sprayed with a decoction of spores obtained from pure cultures according to the method described by Kunkel (7) and Rands (8). At the end of six days they were removed from the moist chambers, wrapped in paper, and kept at room temperature. They were examined again at the end of eight days at which time the following notes were made:

| NUMBER OF FRUIT | CIRCUMFERENCE OF FRUIT | NUMBER OF SPOTS ON EACH FRUIT |
|---|---|---|
| | *inches* | |
| 12 | 6 or more | 0 |
| 1 | 1 | 1 |
| 1 | 1 | 2 |
| 5 | 1 | 6 |
| 3 | 2 | 0 |
| 4 | 2 | 3 |
| 2 | 3 | 0 |
| 5 | 3 | 6 or more |
| 1 | 4 | 0 |
| 2 | 4 | 1 |

Artificial inoculations, in the laboratory as well as in the field, on uninjured fruit have been repeated a large number of times with results similar to those presented above. Positive infection is obtained when the inoculation is made on young, immature fruit approximately less than 6 inches in circumference. A period of from eight to ten days elapses from the time the inoculation is made to the time when the symptoms are first visible.

The results of the artificial infections in the laboratory and field furnish additional indirect proof that the disease cannot originate or spread in transit on mature green fruit of the size which is generally shipped.

*Types of lesions found on fruit and stems, and identity of fungi isolated.* The lesions of early blight on tomato fruit as found in Florida and other Gulf States are small, circular or irregular, brown to gray spots, seldom attaining a size greater than that of an ordinary nail-head, as shown in plate II. Occasionally a much larger spot is produced, but similar in color to the above as shown in plate III, figure A. This has been referred to during the progress of this work as a spreading Macrosporium spot. In Florida this last approximately twenty-five spores being measured at each time. Great variation was found among the individual spores for the same culture. It was also a problem whether the entire length of the spore, including the elongated pedicle or beak, should be measured. It was found that greater variation occurred in this beak than in the body of the spore. In table 5, however, which summarizes the results of these measurements, both are included. The position in the spore where the body ends and the beak begins was necessarily more or less arbitrary; nevertheless, the following points were considered in deciding this question: (a) the light brown tinge of the body, as compared to the hyaline beak, (b) the more uniform diameter of the beak, and (c) the greater distances between the cross septa in the beak as compared to those in the spore body. Examination of the table of measurements (table 5) and comparison of the figures in figure 1 show that the Macrosporiums isolated from typical "nail head" spots vary considerably in size. In addition, there is shown still greater variation in the spores from the isolations from the different lesions. Thus, 18–110 has a length of the spore body, not including the beak, of 47.3 microns, as compared to 79.0 microns for the Macrosporium isolated from New Jersey tomato fruit. Between these two extremes various gradations in size of parasitic Macrosporiums are shown.

Notwithstanding these variations, it is nevertheless evident that the spore measurements likewise separate the Macrosporiums into the same two groups as found when these were studied in culture, or as regards the kind of lesions produced. Those Macrosporiums isolated from typical "nail head" spots from fruit, stems and leaves are invariably smaller in size than *Macrosporium solani* from the potato, or the cultures 19–130 and 19–173, which resemble it.

In addition to the difference in size found in these two groups, the following differences are found, as shown in figure 1. *Macrosporium solani* 19–130 and 19–173, show a large proportion of the beaks forked. In the remainder of the cultures, while a forked beak may occasionally be present, this is an exception rather than the rule. This characteristic of *M. solani* is especially well illustrated by Rands (4) in his figure 4. In addition, the beaks of the spores of *M. solani* from potatoes and Macrosporium cultures from tomatoes resembling it, such as 19–130 and 19–173, are generally coarser, i.e., of larger diameter than those of the Macrosporiums isolated from typical "nail head" spots.

The cultures resulting from isolations made from tomato fruit from Tennessee, Texas, and Louisiana resemble in all respects the cultures obtained from typical "nail head" spots from Florida fruit, which are discussed here.

TABLE 5.

*Measurements of Macrosporium*

| FUNGUS | SOURCE | LENGTH IN MICRONS | | | | | | WIDTH IN MICRONS | | | SEPTA | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Spore body | | | Spore body plus beak | | | | | | | | |
| | | Minimum | Maximum | Average | Minimum | Maximum | Average | Minimum | Maximum | Average | Minimum | Maximum | Average |
| 17–19 | Nail-head spot. Tomato fruit. Florida | 37.8 | 77.7 | 56.0 | 100.8 | 218.4 | 164.4 | 12.6 | 21.0 | 17.8 | 6 | 10 | 7 |
| 17–57 | Potato seedlings. Damping off. Maine | 39.9 | 123.9 | 74.3 | 65.0 | 256.2 | 157.4 | 12.6 | 23.1 | 17.4 | 4 | 14 | 7 |
| 18–110 | Large spreading nail-head spot. Florida | 29.4 | 69.3 | 47.3 | 54.6 | 279.3 | 132.6 | 10.5 | 23.1 | 16.9 | 4 | 9 | 6 |
| 19–130 | Large spot. Ripe tomato. New Jersey | 42.0 | 128.2 | 79.0 | 88.2 | 407.6 | 288.5 | 10.5 | 25.2 | 20.1 | 5 | 13 | 8 |
| 19–134 | Early blight spots. Potato leaves. Maine | 29.4 | 111.3 | 67.0 | 71.4 | 247.8 | 153.1 | 8.4 | 23.1 | 18.2 | 4 | 12 | 7 |
| 19–147 | Nail-head spots. Green tomato. Direct Mounts. Florida | 31.5 | 71.4 | 47.6 | 69.3 | 163.8 | 114.2 | 10.5 | 18.9 | 14.4 | 5 | 9 | 6 |
| 19–148 | Saprophytic Macrosporium. Green tomato. Florida | 21.0 | 42.0 | 31.5 | 31.5 | 75.6 | 52.6 | 8.4 | 18.9 | 12.7 | 4 | 7 | 4 |
| 19–154 | Nail-head spot. Tomato fruit. Florida | 35.7 | 88.2 | 58.7 | 75.6 | 323.4 | 189.3 | 12.6 | 31.5 | 20.7 | 5 | 11 | 7 |
| 19–170 | Nail-head spot. Tomato fruit. Florida | 29.4 | 81.9 | 56.7 | 65.1 | 201.6 | 126.9 | 10.5 | 21.0 | 16.5 | 5 | 12 | 7 |
| 19–173 | Cankers on tomato stems. "Foot Rot." Delaware | 56.7 | 98.7 | 75.1 | 157.5 | 350.7 | 244.1 | 14.7 | 23.1 | 18.0 | 5 | 10 | 8 |
| M. solani | Measurements by Saccardo | | | | 100.0 | 140.0 | | 15.0 | 18.0 | | | | |
| M. tomato | Measurements by Saccardo | | | | 100.0 | 120.0 | | 20.0 | 22.0 | | | | |

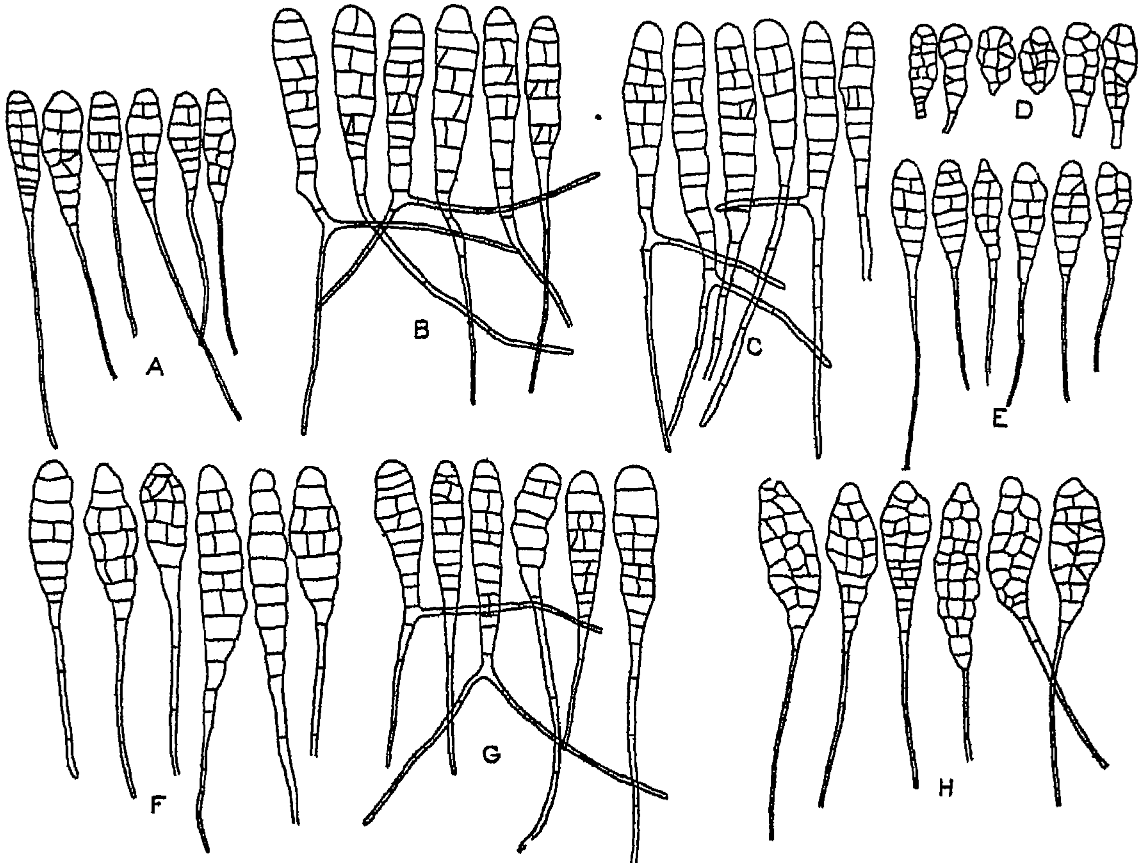

FIG. 1. SPORES OF MACROSPORIUM

A. *M. tomato.* From culture marked 17–19. Isolated from typical "nail head" spot. Tomato fruit from Florida.

B. *M. solani.* From culture marked 19–173. Isolated from tomato plants affected with "foot-rot" from Delaware.

C. *M. solani.* From culture marked 17–57. Isolated from potato seedlings from Maine.

D. Saprophytic Macrosporium. From culture marked 19–148. Isolated from green tomato fruit from Florida.

E. *M. tomato.* From culture marked 18–110. Isolated from spreading "nail head" spot from Florida tomatoes as shown in plate II. Fig. A.

F. *M. solani.* From culture marked 19–134. Isolated from potato leaves from Maine.

G. *M. solani.* From culture marked 19–130. Isolated from tomato fruit rot from New Jersey.

H. *M. tomato.* From culture marked 19–154. Isolated from "nail head" spot from Florida tomatoes. The body of these spores are larger than the average *M. tomato* from "nail head" spots but similar in the terminal beak, in cultural characters, and in pathogenicity.

Consideration of the facts brought out in these comparisons shows without a doubt that the Macrosporiums producing typical "nail head" spots on fruit, stems, and leaves, as represented by cultures marked 17-19, 18-110, 19-154, and 19-170, are different from *M. solani* from potatoes, represented by cultures 17-57 and 19-131. They are also different from 19-130 from New Jersey tomato fruit, and 19-173 from Delaware tomato stems. The latter two cultures agree very closely with *M. solani* from potatoes and should be referred to as such. The above results may possibly explain the apparent discrepancy between the conclusion drawn by Rands (4) and Kunkel (7) as regards the identity of the Macrosporium from tomatoes with *Macrosporium solani* from potatoes. Kunkel (7) refers to the Macrosporium from tomato as *M. tomato.* This specific name is considered a synonym by Jones and Grout (6) for *Alternaria fasciculata*, which is not parasitic on the tomato. The spore measurements as given in the original description by Cooke (10) agree fairly well, however, with the spores obtained from typical "nail head" spots. It is, of course, hard to determine whether Cooke's *M. tomato* is identical with those obtained from "nail head" spots. In view of this fact, however, that it is undoubtedly different and distinct from *M. solani* on potatoes, *M. tomato* Cooke is to be preferred. In the cultures studied here all Macrosporiums isolated from typical "nail head" spots, such as 17-19, 18-110, 1-147, 19-154, and 19-170, should be referred to as *M. tomato*, while 17-57, 19-130, 19-134 and 19-173 should be referred to as *M. solani.*

## SUMMARY

1. Numerous counts made in the field and inoculation experiments in the field and laboratory have shown that infection on tomatoes resulting in typical "nail head" spots takes place without previous injury to the fruit. Such infection, however, takes place only on immature fruit not more than approximately six inches in circumference.

2. Experimental shipments of winter-grown tomatoes from the South, together with artificial inoculation experiments on tomato fruit of various maturities as indicated by size, have shown:

*a.* That the disease commonly referred to as "nail head" spot does not originate in transit on mature fruit picked from an infected field.

*b.* That immature fruit, that is, fruit less than six inches in circumference picked from an infected field and shipped without delay, may develop numerous Macrosporium or "nail head" spots in transit.

*c.* That the disease does not spread from one infected fruit to an adjacent fruit during transit.

3. Isolations made from numerous diseased tomato fruit, stems, and leaves from Florida and other tomato growing sections have shown that the same kind of a lesion is generally produced by the same Macrosporium.

4. Cultural and morphological comparisons of all the Macrosporiums obtained from tomatoes with *Macrosporium solani* E. & M. from potatoes show that:

*a.* Typical "nail head" spots on fruit, stems, and leaves are caused by a Macrosporium different specifically from *Macrosporium solani* from potatoes. For reasons pointed out in the text, the Macrosporium causing such spots should be referred to as *M. tomato* Cooke.

*b.* A stem disease occurring in Delaware and a fruit rot of tomatoes in New Jersey are caused by a Macrosporium which resembles *M. solani* in culture, in morphology and in its pathogenicity. It should therefore be referred to as *M. solani* E. & M.

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PLATE II MACROSPORIUM ("NAIL HEAD") SPOTS ON TOMATO STEMS AND FRUIT

The stem and fruit show typical "nail head" spots as a result of natural infection in a field near Miami, Florida

Rosenbaum Tomato Macrosporium

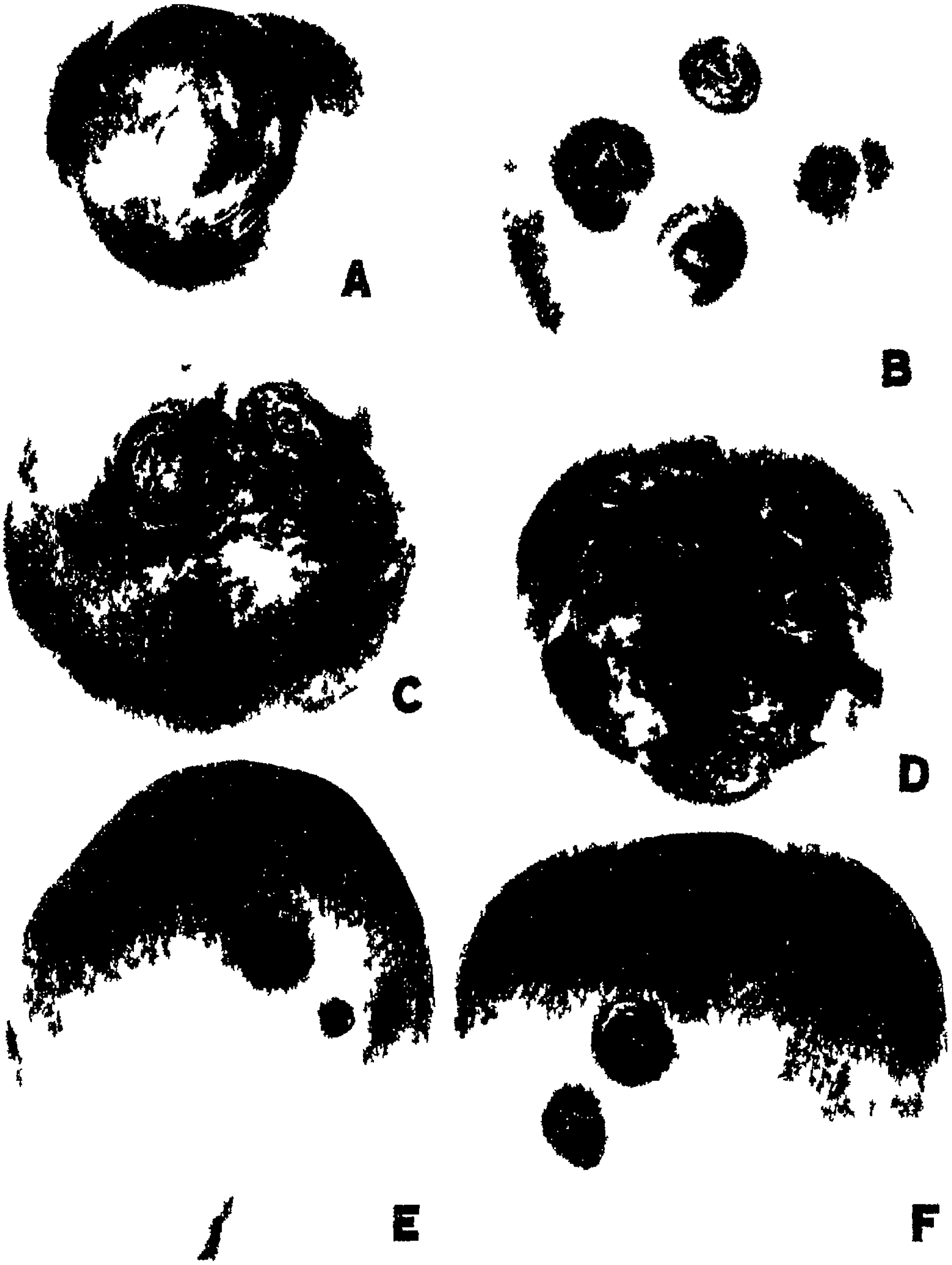
PLATE III. LESIONS ON TOMATO FRUIT DUE TO MACROSPORIUM

Fig. A. Natural infection on ripe tomato showing a spot which has been referred to in the text as a "spreading Macrosporium spot."

Fig. B. Lesions produced artificially by inoculating with Macrosporium from tomato fruit collected in New Jersey.

Fig. C and D. Old Macrosporium spots which show secondary infection by other organisms.

Fig E and F. Typical "nail head" spots on tomato fruit from Florida.



Rosenbaum Tomato Macrosporium

to the right, the callus disappeared, also the dead cells. The path of the solution could no longer be detected. On the right hand side of the section could be seen in the center of a typical xylem tissue a strip of abnormal xylem, consisting of lignified phloem cells mingled with typical xylem ones. The cambium layer was again normal.

## CAUSES STATED

There are two main causes for this development of xylem in the phloem region: (1) The mechanical severance of the vascular tissues; (2) the chemical stimulation of the cells.

### I. THE MECHANICAL SEVERANCE OF THE VASCULAR TISSUES

#### *Review of literature*

Much study has been given to the effect of severing one or more tissues of the vascular system of plants and there is now extensive literature on this phase of experimental morphology. It will be found compiled in Kuster's Pathologische Pflanzenanatomie (1), but it is of value to summarize here at least four of the most important papers, which show the results of mechanical severance of a part of a plant's vascular system.

Hugo de Vries (8) studied the formation of wound wood on a variety of trees. By ingenious methods he succeeded in severing successively the phloem, the cambium, and the newest rings of wood without injury to the other tissues. He found:

*a.* The cells of all tissues formed nearest a transverse wound were shorter than normal ones and lengthened with distance from the wound until they attained their normal length.

Cuts made parallel to a stem axis produced cells of normal length.

*b.* A transverse cut of young wood cells made without injury to the other tissues produced callus and, later, wound wood gradually developed.

*c.* A transverse cut of the cambium alone produced callus and wound wood. In the latter were strands of vessels which connected the normal wood both above and below the wound.

*d.* A transverse cut of the inner phloem made from the inner side (next the cambium) produced wound wood as in the case of the severing of the cambium. But when the cut was made from the outside, however, and the cambium was uninjured, callus was produced.

*e.* Lessening the tension of the bark increased the number of vessels and the diameter of all cells, whether the cut was parallel to the axis of the branch, across it or oblique to it. No wood fibers were formed in the wound wood if the longitudinal tension of the cambium tissue was diminished.

*f.* The nearer the wound, the smaller and more numerous do strands of vessels become. With an increase in the distance of the tissue from the wound the strands of vessels tend to coalesce and finally connect with the normal xylem (see de Vries' illustration, fig. 11, plate 2).

Vochting (7), by transplanting or removing organs of plants, demonstrated the role that the polarity of plants played in the direction which these newly formed strands of vessels took in connecting the severed strands. When the plant was in a normal position the "bridge" or strand of vessels binding the two ends made as short a line as possible. With increasing variation from the usual position of the plant, the directness of the connecting strand diminished.

Simon (5) found by means of ringing or by partly severing the stems of young plants that the first reaction to a transverse wound was the formation of callus, first on the upper, then on the lower side of the wound. Very shortly after this callus formation appeared strands of vessels which were either tracheids or true vessles. In case the wound was made in tissue so young that the cambium had not begun its activity, the vessels appeared before the meristematic cells. These strands formed first near the ends of the severed strands.

In the case of a wound made by transversely severing a portion of the vascular system of a plant, the reunion of the cut vascular tissues proceeded as follows. The strands of vessels formed first on the upper side of the wound at the basal ends of the normal strands of vessels, and then at the apical ends of the strands on the lower side. These vessels often were formed by a direct conversion of cells without previous cell division. A little later a meristematic strand of tissue formed just back of the callus which covered the surface of the wound. In this meristematic tissue vessels appeared, and later the usual phloem and xylem cells. In this manner a "bridge" was formed for the flow of the sap.

Neeff (2) studied the effects of removing the terminal bud from sprouts which were thus forced to continue their length growth from a secondary bud near the tip. He also studied the effects of transverse cuts through parts of the vascular system. Cambium, phloem, and xylem were affected.

*a.* Cambial cells in the neighborhood of the wound divided into short cells. This tissue then proceeded to grow in the direction of the secondary bud, which then replaced the terminal bud. Its cells formed wood and bast parenchyma. It developed vessels and sieve tubes by the absorption of lateral cell walls. These atypical strands sometimes ran at right angles to the original direction of the cells, cutting through medullary rays lying in their path. A cambium layer gradually developed phloem and xylem cells, both groups of elements becoming more normal in shape the farther removed they were from the wound. Transverse wounds of the vascular system of young sprouts produced the same results.

*b*. After the operations, short, dividing cambium cells (Neeff's nomenclature) developed in the phloem region. This tissue developed as described in *a*. In the development of the connecting "bridge" phloem cells were converted without cell division. Bast fibers developed pits. Cells of the medullary rays were changed into short sieve tubes and companion cells. The medullary rays were cut into small parts by these developing strands.

*c*. The xylem region produced a dividing cambial layer which developed abnormal wood. This wood was at first composed of wood parenchyma; later fibers developed. Strands of vessels were found which grew at right angles to the original direction of wood cells. If the wounded xylem tissue was young, the growing cambium layer developed the usual vascular elements for sap transportation. But before these strands were developed, the old wood cells were used in the transportation.

These descriptions formed a picture complete enough to show the processes by which a plant recovered from a mechanical wound which severed a part of its vascular system. There would have been no need for further work had not some experiments made by Schilberszky (4) appeared to disagree with the results described above and apparently agreed with some of those attained by the writer, i.e., the formation of xylem cells on the outer side of the hard bast.

Schilberszky stated that extra fascicular vascular bundles could be formed on the outer side of the hard bast cells in seedlings of *Phaseolus multiflorus* and *P. vulgaris*. This new tissue developed by the activity of the starch sheath or endodermis, which acted as a cambium layer, producing phloem cells on its outer side and xylem cells on its inner. He produced this result by cutting out a part of the seedlings' stems when they were actively growing.

An attempt was made by the writer to repeat Schilberszky's experiments. This could not be done exactly, for the reason that seed of *Phaseolus multiflorus* or *P. vulgaris* could not be obtained at the time the work started. Instead seedlings of the vetch, *Vicia faba*. were used, as were a few seedlings of an edible pea, of castor bean and young shoots of *Polygonum sieboldii*. Schilberzsky's result was not obtained, i.e., the conversion of the endodermis into a cambium layer, such as he figured in figure 5, Plate XXII (4).

### *Experiments with Vetch seedlings*

Results of interest, however, were obtained with vetch seedlings. At a period of rapid growth a segment ½ inch long was removed from the stem of a seedling, leaving but one-half or one-third of the cylindrical stem through which the sap could flow. This remaining segment of stem responded by forming a callus over the cut surface. Behind this callus formed a meristematic layer connected with the cambium of the old vascular system. This layer in time produced phloem and xylem cells and thus reformed as nearly as possible the original circular vascular system. During this time the vascular elements left in the segment of stem increased in quantity by secondary growth. These results were duplicated with seedlings of pea and castor oil bean, and with shoots of Polygonum.

The nearest approach to Schilberszky's results was obtained when so large a segment of the seedling's stem was removed that the wound was next to the xylem cells and few of any pitch cells were left. In this case a meristematic layer of cells formed just outside the hard bast just as is pictured in Schilberszky's figure 3, plate XXII (4). But this meristematic layer, as Schilberszky apparently failed to see, was connected with the original cambium layer. Later some vessels developed in this new formed meristematic layer but at right angles to the normal direction of the cells. Serial transverse sections of such stem segments demonstrated this connection of the meristematic and newly formed vessels with the old vascular system. They could be seen passing between vascular bundles to the position outside the hard bast cells. The experiments did not show that the endodermis or the region about it was, in and of itself, especially active in growth, but that all the cells in the vetch seedlings' stems, except the hard bast cells, were capable of growth when they lay near a wound.

## *Summary*

When there was a mechanical severance of growing vascular tissues (and here no chemical was introduced), new passages for the sap were formed invariably. When the plant was in a normal position such new passages always connected the severed ends and took a nearly direct route.

These conclusions are based on experiments of de Vries, Vöchting, Simon, and Neeff, and have been confirmed by the writer. The only one of the authors here considered who did not agree with this statement was Schilberszky. His observation that there were new formed vessels outside the hard bast region was right, though only in such cases where so large a section of the seedling's stem was removed that little if any pitch cells were left. However he failed to see the connection between these vessels outside the hard bast region and the normal vessels, and so did not realize the part they played in the transportation of the sap.

### *Condition of injected trees*

When comparing the results obtained from cutting with a knife through a part of the vascular system of a plant with those obtained by injecting it with chemicals, it should be remembered that these results varied, because the effect of the chemicals varied and also because there was a variation in the effect of a solution in an injected tree due to the retention of the chemical by the cell walls and the consequent increasing dilution of the passing stream.

An examination of the trees showed:

*a*. If the solution killed, but did not spread rapidly through the tree, then a splitting of the bark occurred along the path of the solution and a typical callus formed such as would have developed had the bark been cut by a knife. The more toxic the solution the more normal the wound tissue.

Sometimes a hole was worn in the young wood by the passing solution. If the cells surrounding the hole were alive the hole was filled by a callus growth.

*b*. If the solution was less toxic than the one described in *a*, it stopped the growth of the new wood, the cambium and inner bast, either one, two or all three of the tissues, according to its toxicity and to the path of the solution. Here wound wood formed which connected the severed or blocked vascular tissues. But instead of wound tissues such as formed about a cut, wound tissue formed in a narrow area extending up and down the tree, often for a number of feet.

*c*. If the solution did not injure or inhibit cell growth, it caused an irregular growth of cells. This showed itself in the phloem region in atypical lignified cells formed in irregular strands which connected with the normal xylem tissue. This third phase of the results from tree injection was caused by chemical stimulation.

## II. CHEMICAL STIMULATION OF CELLS

### *Smith's experiments with chemicals*

The paper "Mechanism of Tumor Growth in Crown Gall" recently published by Erwin F. Smith (6) showed to what an extent the presence of certain chemicals could excite plant cells. His figures (6) on plates 56, 58, 59, and 61 showed these effects most clearly. They showed intumescences produced on the under surface of cauliflower leaves when exposed to the vapor of a mixture of ethyl alcohol and acetic acid. Here there could be no question of a severance or blocking of the original vascular system of the plant, by a bruise, or a cut. Chemical irritation at a single point on the leaf had continued long enough to produce cell growth and division. This activity had been followed by the formation of conducting tissue connecting the growing region with the normal vascular system. In Smith's illustrations, figures 3, 4, and 5, plate 58, showed transverse sections of these intumescences and figure 1, plate 61, a longitudinal section, in which incipient vessels could be seen entering the region of actively growing cells.

The extent to which chemical irritation could produce extra vascular tissue was shown by Smith's illustrations on plates 37, 38, 39, 40 and 41, where the injection of a 5 per cent solution of monobasic ammonium phosphate into an internode of a castor bean seedling had caused the formation of a second vascular system in the pith cells lining the hollow internode.

### *Experiments with Polygonum and Vetch seedlings*

The writer produced a result similar to Smith's with the castor bean seedling, but less fully developed, by exposing the cavities of internodes of young shoots of *Polygonum sieboldii* to the stimulation of air. This was done by removing a segment of stem from an internode. The exposed pith cells lining the cavity formed a callus. Behind this developed a meristematic layer in which vessels appeared. These vessels connected with xylem cells in the normal vascular system.

In order to compare more readily the difference in results between mechanical severance of the vascular tissues and that produced by the injection of chemical solutions, experiments were made with vetch seedlings. During a period of rapid elongation, these were injected by means of a hypodermic syringe. The solutions of chemicals (circa 1 cc.) were introduced in the internodes with as little injury to the stems as possible. When examining the injected plants the region around the place of injection was discarded. The following results were obtained:

*a.* When the solution killed the tissues a meristematic tissue cut off the dead cells from the living. If part of the vascular tissue was killed new tissue developed. (Example: mercuric bichloride 1/500 G. M.).

*b.* When the solution inhibited cell growth (near the place of injection), the result was the formation of a meristematic layer, and (1) cells at a distance showed little if any response (picric acid 1/10000 G. M.); (2) cells at a distance showed irregular division (example: thymol 1/1000 G. M. and to a much greater extent cell division was produced by sodium hydroxide 1/50 G. M., lithium nitrate 1/50 G. M. and Knop's solution).

*c.* When the solution produced no effect (distilled water).

These effects obtained in the vetch seedlings were very irregular compared with those produced with the knife.

In the results described under *b*, the cells in the cortex and pith regions divided once, twice and sometimes three times and generally showed large nuclei. No vessels were noticed among these dividing cells. The cells of the vascular bundles were unchanged, but many of the cells in the neighborhood of the cambium contained unusually large nuclei. This especially was the case in those stems injected with Knop's solution. These effects produced in the seedlings by the small amount of chemical solution injected, about 1 cc. in a plant, were regarded as a first phase of cell response to the irritation caused by the presence of a chemical.

When there was a chemical stimulation of plant cells, they responded by cell division and sometimes growth. In case the stimulation was sufficiently pronounced, the resulting region of actively dividing cells developed vessels which connected with the normal vascular system of the plant.

With continued stimulation more fully developed xylem tissue formed.

### *Examination of injected tree trunks*

An examination of injected trees was made by sawing the trunks into sections and then following the path of the solution up and down the tree from the injection hole to the point where phloem, cambium, and xylem regions were normal in appearance. Blocks of wood were removed from these sections at varying distances from the injection hole, both in the path of the solution and at the side. Microscopical slides were prepared from these blocks.

These slides showed that the greatest amount of extra xylem, i.e., xylem in the phloem region, was in the neighborhood of the injection hole and the killed tissue extending up and down the tree from the hole. In general the extra xylem cells in this region were normal in shape and developed in numerous strands of uniform cells which ultimately united with the normal vascular tissue. See fig. 1, plate IV toward the side marked +. Figure 1, plate V shows an enlargement of a part of a cross section of a strand or bridge of xylem (as in fig. 1, plate IV, showing the development of normally shaped xylem cells on the outer side (nearer the bark) of a row of bast fiber cells. On the inner side of the bast fiber row the walls of the medullary ray cells are partially lignified, but have not changed their size and shape.

The slides also showed the presence of extra xylem cells of normal shape and small size in the phloem region when the cells of the regular xylem were normal in appearance, where no sign of an occlusion of the tissue was visible and where the place itself was so far removed from killed tissue there could be no question of effect from severed vascular tissues. Figure 2, plate V shows a longitudinal radial section of such a region.

In this region there also were extra xylem cells which were phloem cells with lignified walls, also usually arranged in irregular strands. Frequently in these xylem strands there were groups of phloem cells with intercellular species in their midst. This pathological tissue connected with the normal vascular system of the tree. Such tissue can be seen in figure 1, plate IV on the side marked 0. It can also be seen in the center of figure 2, plate IV.

In the great majority of cases the injected trunks showed that this region containing irregular or extra xylem was, comparatively speaking, a small one. Usually pathological tissues could not be seen more than two feet from the injection hole. This was partly due to the fact that when the trees were injected an endeavor was made not to kill them by the treatment and consequently the chemical solutions used were dilute.

## SUMMARY

The causes for the production of pathological xylem in the phloem region of the trunks of injected chestnut trees is two-fold: (1) The mechanical severance of vascular tissues; (2) the chemical stimulation of cells.

1. The xylem is caused by a mechanical block formed in the neighborhood of the wound; in such a region meristematic cells and vessels arise. The vessels develop from new meristematic cells and from local cells. Strands of such meristematic cells and vessels connect the severed ends of the vascular tissues. The above mentioned facts have been brought out by the experiments of de Vries, Vöchting, Simon, and Neeff, and have been confirmed by the writer's experiments with seedlings.

2. The xylem, due to a chemical stimulation, forms in the neighborhood of cells which have begun dividing and connects that growing region with the regular vascular system. This procedure of cell growth was brought out by Smith's results from subjecting cauliflower leaves to chemical vapors and by the writer's from exposing the interior of young Polygonum internodes to the air.

The writer's examination of tree trunks injected with various chemicals shows that frequently the two factors, mechanical severance of vascular tissue and chemical stimulation, act simultaneously in the formation of strands of pathological xylem in the phloem region. Observation, however, also shows that a mechanical obstruction in the vascular tissues results in a more regular formation of strands of xylem cells than when they are caused by chemical stimulation.

The writer finds that at a marked distance beyond injured or discolored tissues, irregular strands of xylem are found in the phloem region, strands composed of irregularly formed cells and of irregular formation. This formation is not caused by a severance of a part of the tree's vascular tissues, but by a chemical stimulation.

INVESTIGATIONS IN FOREST PATHOLOGY
BUREAU OF PLANT INDUSTRY,
UNITED STATES DEPARTMENT OF AGRICULTURE

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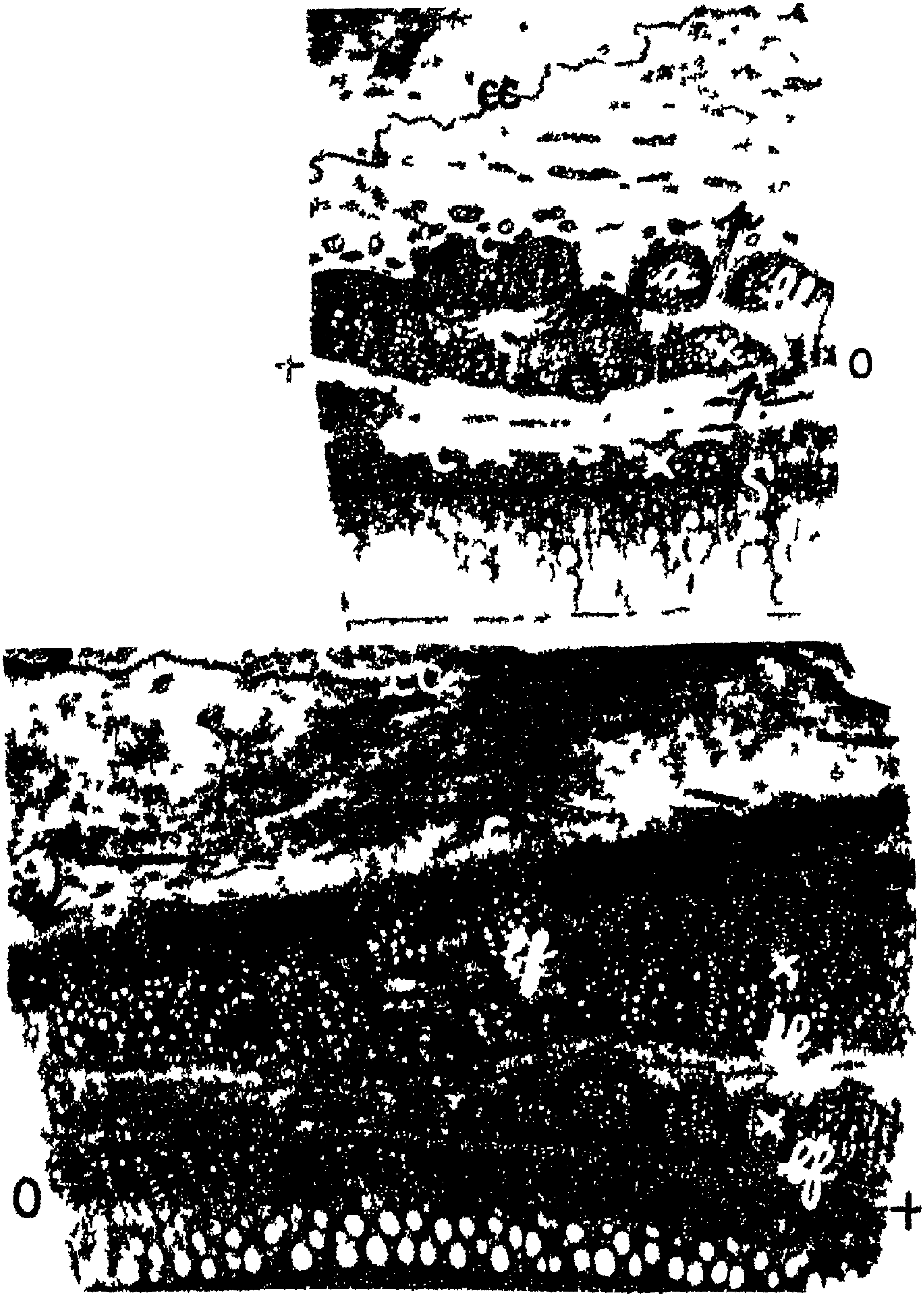
## PLATE IV

FIG. 1. Tree no 21C, injected with lithium carbonate, 1/500 G. M. Cross section of bark and wood *p*, phloem; *x*, xylem; *c*, cambium; *cc*, cork cambium or bark cambium; *bf*, bast fibers; *a*, extra strand or bridge of xylem in the phloem region; s, stain caused by the injected lithium salt. (See text p. 24.)

FIG. 2. Tree no. 143E. Injected with nitro phenol para 1/1000 G. M. Cross section of bark and wood. *p*, phloem; *x*, xylem; *c*, cambium; *bf*, bast fibers. (See text p. 24.)

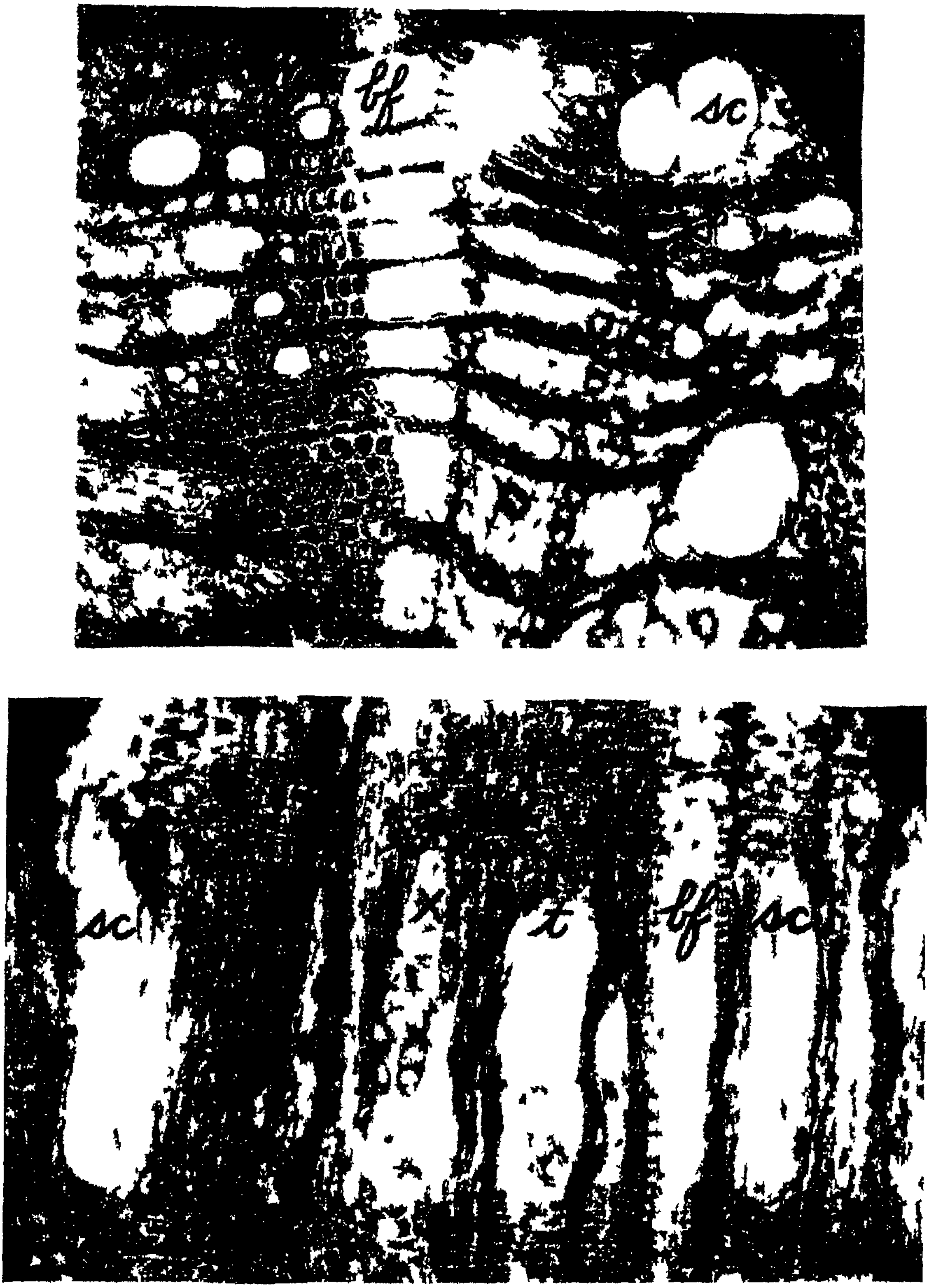


RUMBOLD PATHOLOGICAL XYLEM

## PLATE V

FIG. 1 Enlargement of strand of wood in the phloem region marked *a* in figure 1, plate IV. The inner region of the strand of xylem, showing the development of zylem cells on the outer side of a row of bast fiber cells. The large openings in the xylem are tracheae. *x*, xylem; *bf*, bast fiber cells; *mr*, medullary ray cells; *sc*, stone cells.

FIG. 2. Tree no. 21C. Longitudinal radial section of strand of xylem in phloem region. The left of the picture is nearest to the outer bark, the right of the picture nearest the wood rings. *sc*, stone cell; *x*, xylem cell; *t*, trachea; *bf*, bast fibers.



RUMBOLD PATHOLOGICAL XYLEM

# THE MOSAIC DISEASE OF THE SOLANACEAE, ITS RELATION TO THE PHLOEM-NECROSIS, AND ITS EFFECT UPON POTATO CULTURE

H. M. QUANJER

WITH FOURTEEN FIGURES IN THE TEXT

The "curl" of potatoes is an old disease which hitherto has been connected by earlier authors with degeneration or senility of the variety. In the beginning of this century Dr. O. Appel made it clear that the term "curl" has been applied to a number of distinct diseases; he set apart a particular form which he called "leaf roll;" for another type he retained the old name "leaf curl" (Krauselkrankheit). This last type is the same which is called "curly dwarf" in America. Another form related to this group is described in America as "mosaic" (Orton, 1914).

The first requisite for experiments on the contagiousness of these diseases is a great quantity of reliable healthy plants. Since these diseases are widely spread in the fields it is necessary to cultivate healthy plants on a large scale. It is the merit of Oortwyn Botjes to have detected the mode of dissemination in the field and to have based on it a method of cultivating disease-free plants.

The writer has studied the histological and physiological side of the problem. He detected phloem-necrosis in 1908 and described it for the first time in 1913, after having made observations on it in strains of potatoes of the variety Paul Krüger and some other varieties cultivated during five years. He has come to the conclusion that the old conception of "leaf curl" contains two distinct diseases.

1. Leaf-roll, which may better be termed phloem-necrosis or, according to Pethybridge, lepto-necrosis. It is contagious and pseudo-hereditary (Quanjer, V. D. Lek, and Oortwyn Botjes, 1916), and by means of these peculiarities distinguishable from pseudo-leafroll caused by soil influences. Moreover, it is characterized by necrosis of the phloem strands and is thereby discernible from foot and wilt diseases (Quanjer, 1913).

That the phloem cells are affected in cases of leaf-roll is confirmed by Dr. E. Artschwager (1918), though he believes there are exceptions. The writer is convinced, as far as the variety Paul Krüger is concerned, that phloem-necrosis is the only internal symptom which distinguishes true leaf-roll from a number of other pathological conditions which have some resemblance to it and which might have played a part in Artschwager's one year's observations.

2. Leaf-curl (curly dwarf). This may be called more appropriately mosaic, as it is the intense form of the latter disorder. In several varieties of potatoes Oortwyn Botjes and Quanjer have observed that healthy plants exposed to contamination by neighboring mosaic or curly dwarf plants show in their progeny the first faint symptoms of mosaic, which accumulate and pass into the well-known curling and dwarfing in the following generation (figs. 4 and 5).

There is a good deal of analogy between phloem-necrosis and mosaic. These diseases are not discernible, or if so very slightly, in the year in which infection occurs. In the case of phloem-necrosis sometimes and in some varieties it may be recognized in a mild form in the upper parts of the plants as "primary leaf-roll;" in the progeny of the primarily infected plants the disease develops in its intense or "secondary" form, which admits of a reliable diagnosis. The same thing holds good with regard to the mosaic disease. In a primarily infected plant the symptoms are not discernible to an appreciable degree; even in the direct progeny it may vary from a scarcely discernible form to intense mosaic; in the following generation the mosaic may be coupled with a curled or dwarfed habit. In very sheltered places the more intense form does not develop,

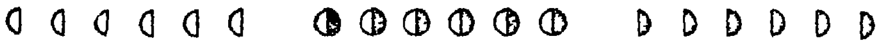


FIG. 1. At the left, six halves of healthy tubers, the other half of each of which has been transplanted to a half diseased tuber (in the center). At the right, the six control halves of the diseased tubers. The transplanted halves grow into one by means of the phloem portions of their vascular rings. After being planted the shoots of the sound halves will prove to be contaminated.

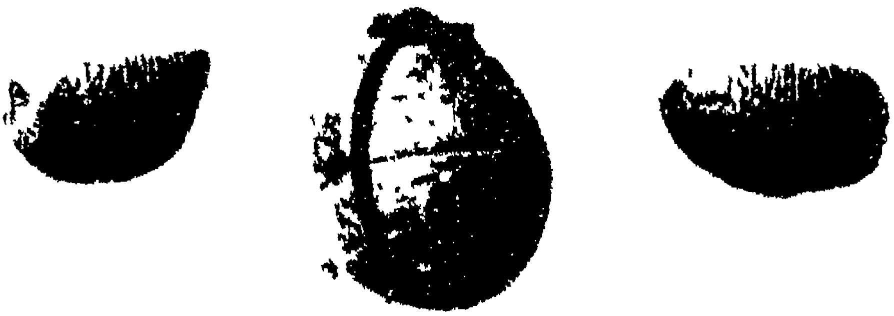


FIG. 2. In the center is shown a combination of a sound half and a diseased one; the other halves are to be found at the left and right.

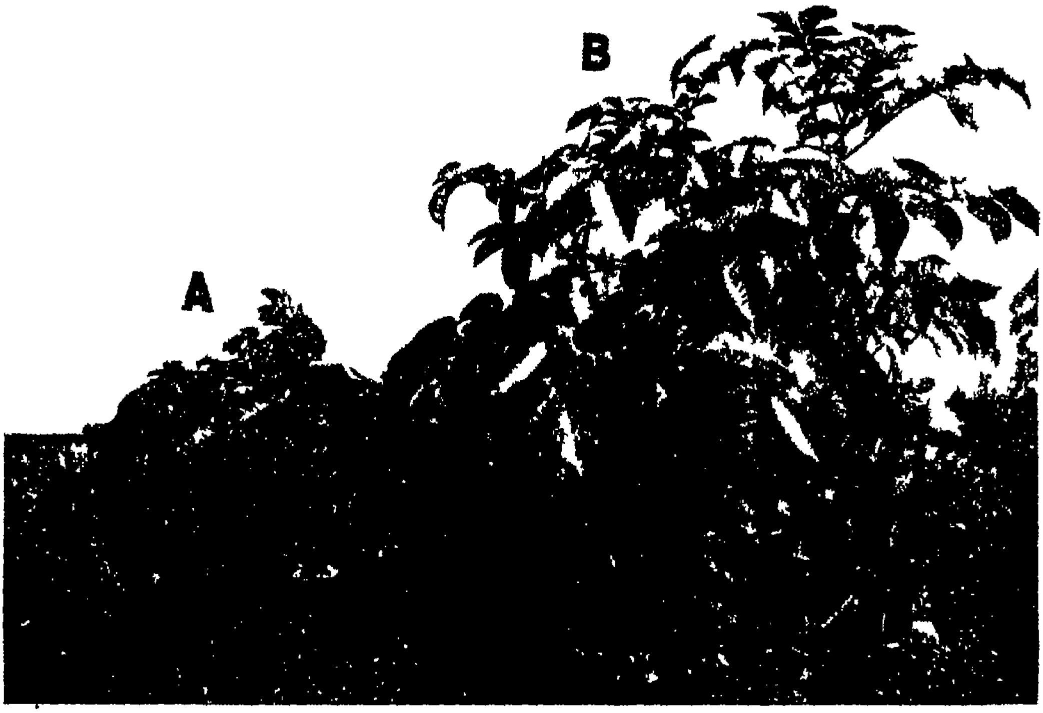


FIG 4 *A* PLANT OF VARIETY EIGENHEIMER WITH THE CURLY DWARF FORM OF MOSAIC *B* A HEALTHY PLANT OF THIS VARIETY

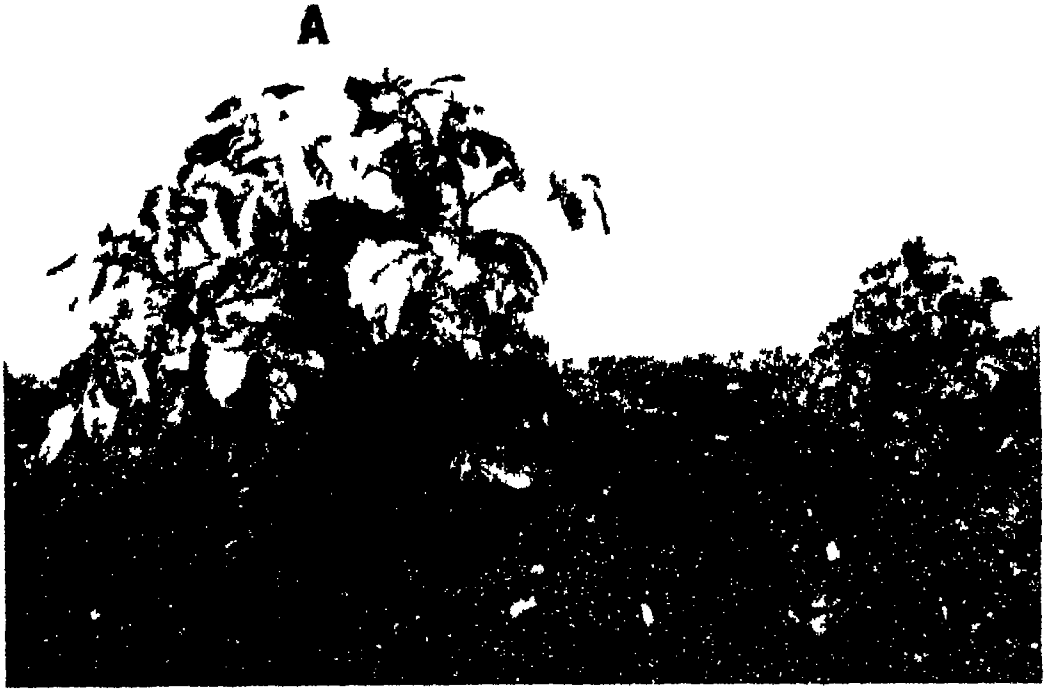


FIG 5 *A* PLANT OF VARIETY EIGENHEIMER WITH THE MILD OR MOSAIC FORM OF DISEASE *B* PLANT OF THIS VARIETY WITH THE CURLY DWARF FORM

as is the case with phloem-necrosis. So the progress of mosaic in several varieties is slower than that of phloem-necrosis.

That both phloem-necrosis and mosaic are contagious can be proved by grafting diseased scions on healthy stocks or by transplanting halves of diseased tubers on halves of healthy ones (figs. 1 and 2). The same methods have been used in order to prove that different forms of mosaic, varying according to the variety of potatoes, are indeed the very same disorder.

The chief sources of infection by phloem-necrosis and mosaic in the field are neighboring plants (fig. 3), (Oortwyn Botjes 1916). In figure 3 the upper row of circlets represents a row of plants growing in 1917 on heavy clay, being the progeny of one plant. This row was planted in an isolated place, so that infection could originate only from diseased plants in the row itself. The two blackened circlets are plants attacked by phloem-necrosis, the six gray ones by mosaic. Of one of these (the sixth), a portion of the stalks are mosaic. Under each of the circlets a vertical row of five circlets is drawn, representing the asexual progeny in 1918 of the corresponding plant in 1917. It is seen from the progeny of 1918 that phloem-necrosis has spread in 1917 to the next neighbor and has infected even the following neighbor so slightly that the disease is appreciable only in some sprouts of one plant of its progeny. The extension of mosaic to the neighboring plants occurred in an entirely analogous way; the partially infected plant has an entirely diseased offspring. Generally the mosaic proceeds to the next neighbor; in exceptional cases the following neighbor also is infected. On heavy clay soil only the very nearest neighboring plant was infected. On light sandy or peaty soils the infection passed on to the third, fourth, or fifth plant, or to a distance of 2 or 3 yards. In an experimental way it has been shown that as a rule the infection passes through the soil, not through the air. This is illustrated by figure 6, where the two vertical rows of circlets represent each a row of five mosaic plants of the variety Paul Krüger, growing on

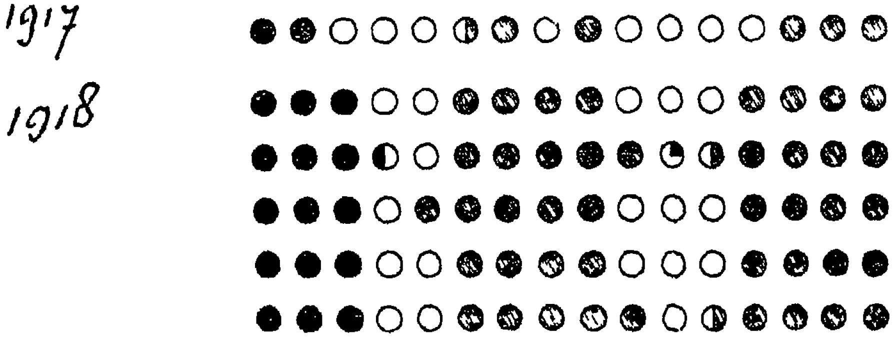


FIG. 3. DIAGRAM ILLUSTRATING FIELD SPREAD OF MOSAIC AND PHLOEM-NECROSIS

sandy soil; the two horizontal rows are healthy plants of the variety Bravo. In the left plot an iron partition was fixed into the soil to a depth of above one yard; in the other plot (situated at some distance) the roots of the healthy and diseased plants could intertwine. The next year the whole progeny of the Bravo of the left plot was still healthy; the whole offspring of the Bravo of the right plot was diseased.

In 1915 a row of tubers attacked by phloem-necrosis and a row of healthy ones was planted side by side (at the left) (fig. 7). Between them and above the ground a double wire was erected so that the roots could reach each other but the vines could not. On another plot (at the right) a row of tubers attacked by the same disease was planted at the side of a trench in which healthy plants were placed in pots. Each pot was wrapped in "wood-wool," so that the roots could not reach those of the diseased plants; the vines, however, could touch each other. In 1916 all the healthy plants of the first plot had a diseased progeny, whereas all the pot plants of the other plot had healthy offspring.

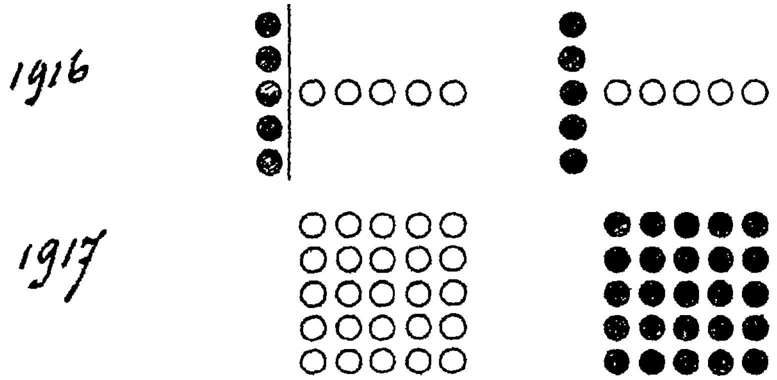


FIG 6. SPREAD OF POTATO MOSAIC THROUGH THE SOIL

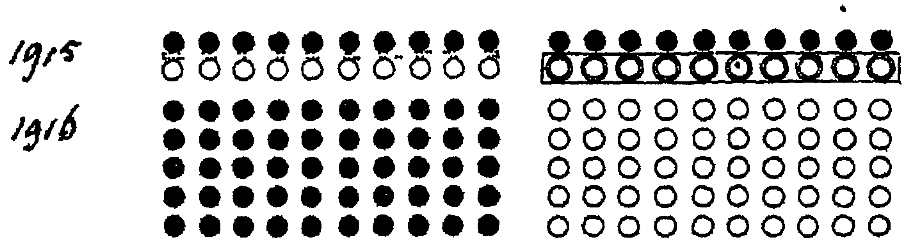


FIG 7 SPREAD OF POTATO PHLOEM-NECROSIS THROUGH THE SOIL

In the experiment illustrated in figure 8, cells, zinc below, glass above, contained two potato plants, a healthy one and a leaf-rolled one, growing side by side. In the first two cells the roots could join but the "vines" were separated by a glass partition. In the following two cells the roots were separated by pots so that the foliage could touch. In the last two cells both roots and vines were separated. The result in 1916 was that contamination had occurred only where the roots could intertwine.

In figure 9 is illustrated the same experiment on mosaic. In each pair of cells the varieties "Blauwe" and "Eigenheimer" have been alternately used as source of infection. The healthy plants, which were exposed to infection, were of the variety Eigenheimer. No matter which variety was used as the source of contamination, the Eigenheimer in every case caught the disease where the roots were exposed to it; it did not where the foliage was exposed. From this it is clear that the mosaic of either variety is identical. When in 1917 the Eigenheimer used as source of infection had the mild or "mosaic" form of disease, in 1918 it had developed into the "curly dwarf" form (indicated by a darker gray color). The newly infected plant had the weak mosaic in 1918. So the progression from mosaic to "curly dwarf" has been proved. When these experiments were repeated in 1918 the result was the same as concerns subsoil in-

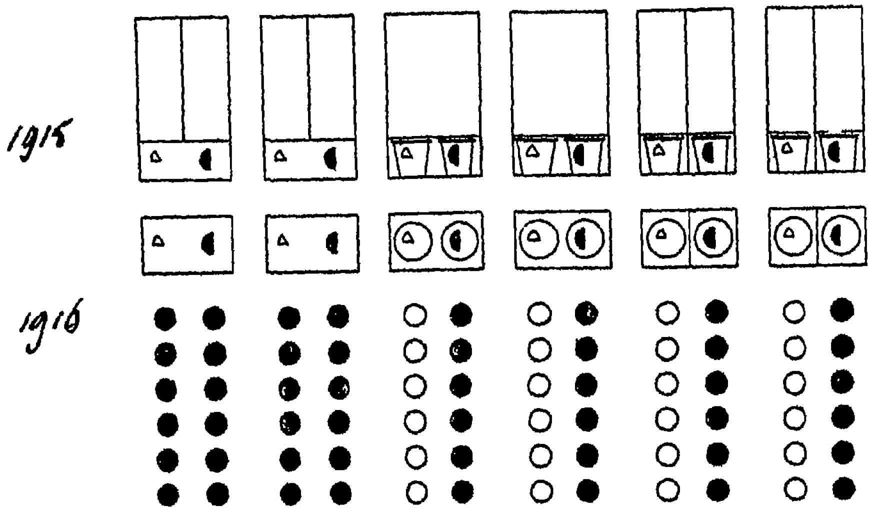


FIG. 8. SPREAD OF POTATO LEAF-ROLL

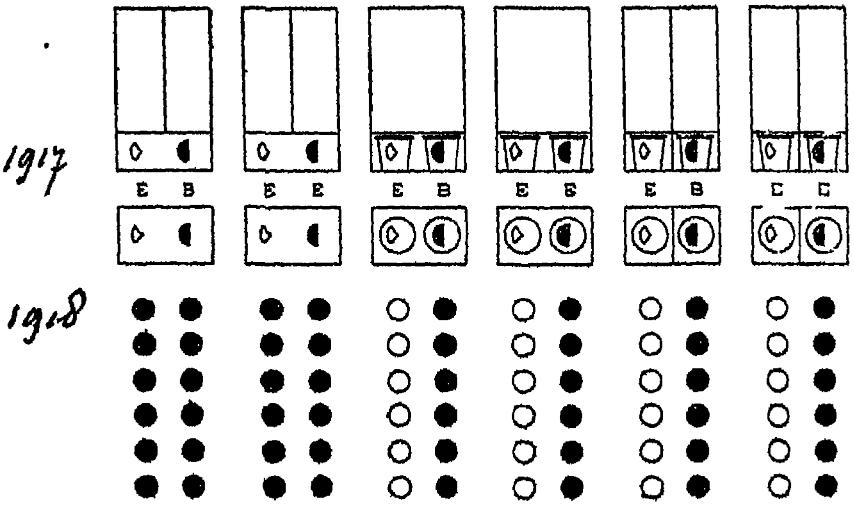


FIG. 9. SPREAD OF POTATO MOSAIC

fection, but some of the plants which were exposed to above ground infection only, also caught the disease as was to be seen from their progeny in 1919. The variety Blauwe is not able to assume the "curly dwarf" form. Although it may be severely diseased, the disorder will still be expressed as mosaic.

The question arises whether the contagia (virus or more probably ultramicroscopic parasites) causing these diseases can live in a saprophytic condition in the soil from the moment that it has been deposited there by diseased plants till the time when a new crop of potatoes is raised. The experiments undertaken to solve this question at Wageningen, in the southern part of our country, have not led to any uniform results in the case of phloem-necrosis. It is to be borne in mind that it is very difficult to determine whether primary infection has occurred by observing the mild symptoms of this disease in the upper parts of the plant, as the latter may be easily confounded with injury due to soil conditions or to attack of the subterranean parts by various parasites. Therefore not only the plants of the experiment itself, but also the progeny of each of them has always been observed. The writer's first experiment in 1915 on soil infection with phloem-necrosis, on plots which had borne diseased plants some years previous, led to positive results. Control plants on plots where potatoes were never grown kept healthy, but when these experiments were repeated in 1917, after a very severe winter had killed all living parts of the potato plant to a great depth, the existence of soil infection could not be proved. Oortwyn Botjes in the north of our country has, from 1913 to 1918, made several experiments on soil infec-

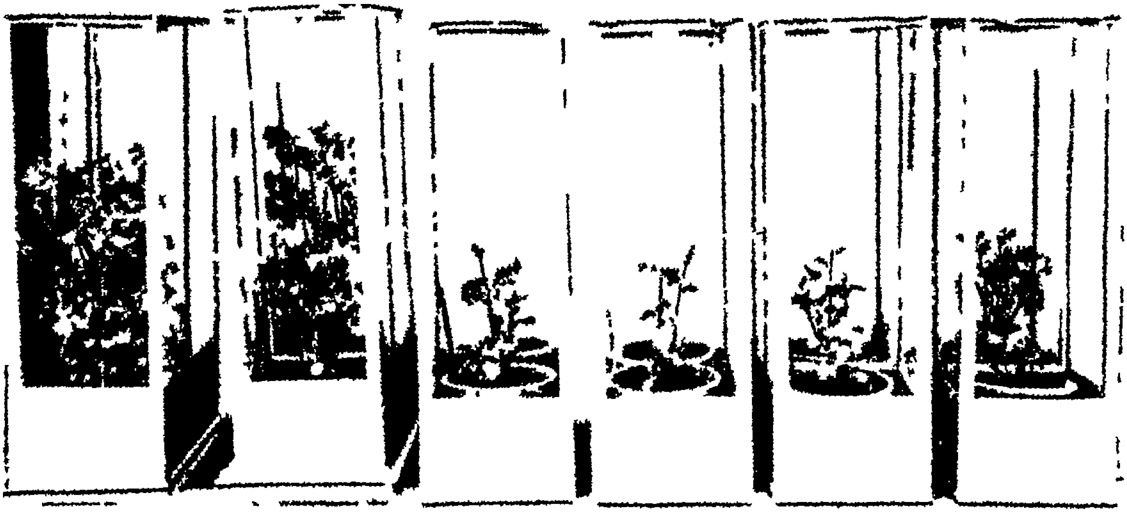


FIG. 10. PHOTOGRAPH OF THE CELLS REFERRED TO IN FIGURES 8 AND 9

tion and never saw traces of it, either in the plants of the experiment or in their progeny. The number of intervening winters during the term of his experiments varied from two to five. Wortley (1918) agrees with Oortwyn Botjes in this respect. The following circumstance is to be considered in explaining the positive result of 1915. It is observed that in Wageningen, in mild winters, some deep grown tubers often remain alive in the soil, from which stunted plants develop, which, even when the field is regularly cultivated, may again produce some deep growing little tubers. As shown in figure 11, tubers are found also at a depth of about half a yard in the soil, which, after having passed the first winter, produce no stalks but only tuber-bearing stolons (cf. also Vochting, 1902). That the contagium lives as a saprophyte in the soil is not probable; it seems to depend upon living parts of the plant. It is scarcely to be supposed that the contagium could keep in this way for more than two winters. The observations of Oortwyn Botjes (1919) give occasion to the suggestion that some of the infections ascribed by the writer to the soil in reality originate from diseased plants which were growing at a distance of about twenty yards from his experimental plots.

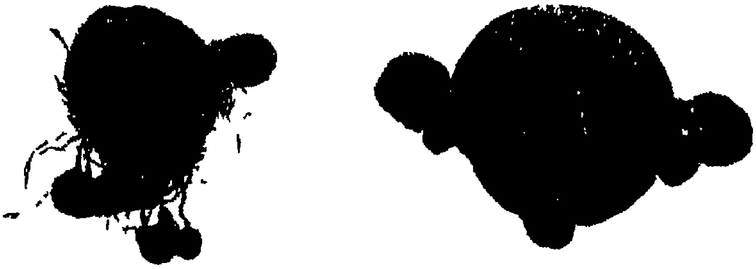


FIG. 11. DEEPLY BURIED POTATOES, SHOWING TUBER FORMATION WITHOUT FOLIAGE

In experiments on soil infection with mosaic negative results were obtained in 1917-1918.

As the contamination of neighboring plants in the case of both diseases passes through the soil and the growing together into an organic union of the tissues of the roots is not known to occur, it would seem that the contagium lives for some time in the soil until it is taken by the roots. It is a matter of doubt whether wounds are essential for the catching of the infection.

Tobacco mosaic is identical with or at least closely related to potato mosaic. Tops of mosaic potato plants have been grafted on healthy tomato and tobacco, so have tops of mosaic tomato on healthy potato and tobacco, and tops of mosaic tobacco on healthy potato and tomato. It was stated that the disease always proceeded from tobacco to tomato and reciprocally in about two weeks. Spreading from tobacco to potato or vice versa has not yet been observed by the author, but positive results were obtained in the grafting of tomato on potato of the variety "Zeeuwsche Blauwe" and back to tomato. With two other potato varieties, Eigenheimer and Bravo, no interchange occurred. So this question has as yet been only partially solved. It would seem that the contagium of tobacco has more difficulty in adapting itself to potato than to other Solanaceae, and a wide field opens here for experiments on adaptation. At all events, it is clear now that the name mosaic is to be preferred for the potato disease and that the term "curl" has only an historical value.

In the case of potato leaf-roll and mosaic there is some irregularity in the infection of neighboring plants, some of them giving a progeny which consists partly of typically diseased and partly of healthy plants. Also in primarily infected plants, and even in secondarily leaf-rolled plants, not all sieve strands are attacked. This would make us consider the contagium rather as a parasite than as a fluid. The same consideration holds good for the infectious mosaic of ornamental Abutilons and other Malvaceae. When this beautiful variegation is propagated by grafting on the perennial Malvaceous plant *Sida napeae*, some of the shoots will show the symptoms of mosaic next year, whereas others are exempt from it (Lindemuth, 1907). Phloem-necrosis of coffee (Stahel, 1917), yellow-stripe and sereh of sugar cane in Java, which also have been considered as degeneration diseases, mosaic of beet (Lind, 1915), peach yellows and rosette (E. F. Smith, 1891), the spike disease of sandal (Leslie C. Coleman, Mysore, Bangalore), and the Japanese mulberry disease resemble very much the potato diseases described here. Schizo Miyake of the Imperial Agricultural Institute, Nakano, Japan, will soon publish his investigations, from which it will be clear that this mulberry disease is related to leaf-roll with respect to the phloem being attacked and to infectiousness.

Only the new leaves and axillary shoots are able to assume the symptoms of mosaic and related diseases; in other words the contagium once introduced into the plant is directed with the flow of organic material to the nascent tissues. The fact that this flow passes through the phloem strands is most conspicuous in the case of phloem-necrosis, where the conducting tissues themselves have been attacked already. How the transport of starch from the leaves downward is prohibited by the phloem-necrosis is shown in figures 12, 13, and 14,[1] which give sketches of healthy and diseased plants that were submitted in the very early morning to

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[1] Quanjer, H. M. Sur la fonction du tissu criblé. *In* Meded. Wageningen. Landbouwhoogeschool, 16: 95–104, fig. 3, 4, and 5. 1919.

Sach's reaction with iodine. Healthy plants have conveyed all their carbohydrates to the tubers, but the lower leaves of the secondary diseased ones are still full of starch. In this type of plant phloem-necrosis proceeds from the base upward as the external symptoms do and likewise the stoppage in the translocation of starch is most efficacious in the basal leaves. In the primarily diseased plants a little phloem-necrosis is found in the upper part of the stalk and the upper leaves. Here the starch translocation is inhibited, whereas the lower leaves are empty. The

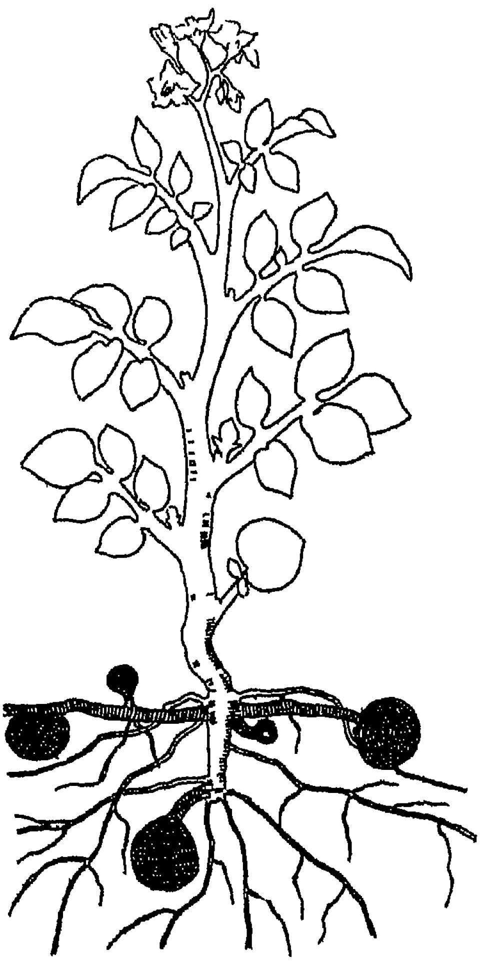

FIG. 12

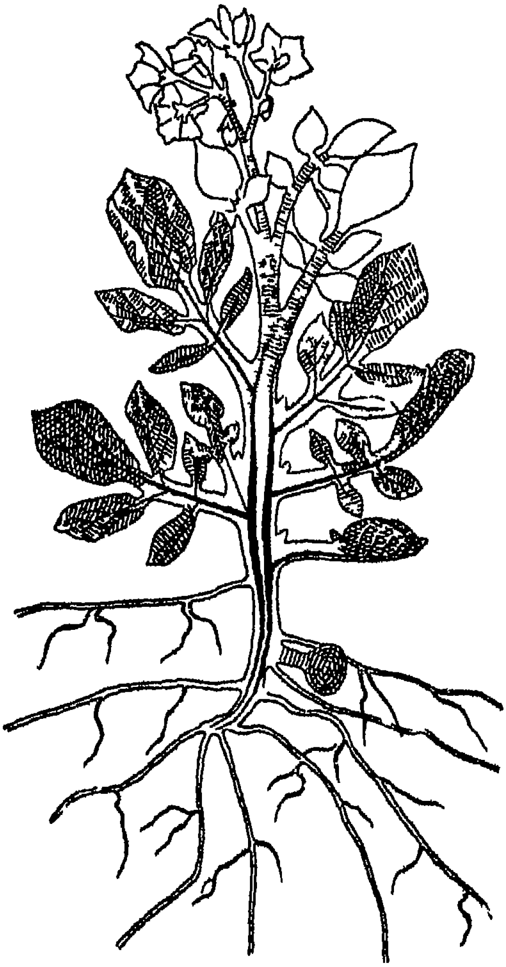

FIG. 13

FIG. 12. DISTRIBUTION OF STARCH IN THE EARLY MORNING IN HEALTY POTATO PLANT

It is to be met with only in the tubers

FIG. 13. SIMILAR EXPERIMENT IN A PLANT WITH SECONDARY LEAF-ROLL

The necrosis of the phloem strands, as pointed out by the black lines in the stalk, is most intense at the base. Here the translocation of the starch has almost entirely come to a stop, as can be shown by means of the iodine reaction of Sachs, and is indicated in the drawing by a black shading.

stalk, because of the delay in the transport, has not been able to convey all its starch to the tubers.

In physiology, the old dispute between Hanstein (1860), who supposes the carbohydrates to pass through the phloem strands, and Sachs (1863), who supposes them to pass through the parenchyma, has never been settled. The author's observations are strongly in favor of Hanstein. Only the fact that the transport of starch from the light colored parts of the leaves of mosaic potato and tobacco plants is delayed, even without phloem-necrosis being present, gives rise to the suggestion that some enzymatic disturbance may also take part in the slackened translocation of organic food. But the tardiness in the translocation of starch in the diseased areas of mosaic leaves is much less than in the leaves of plants suffering from phloem-necrosis.

Whereas the transmission of either phloem-necrosis or mosaic to the progeny of diseased potato plants by the tubers takes place without an exception, the transmission by way of the embryo is rather rare; in the case of tomato mosaic it sometimes occurs; in the case of tobacco mosaic it is not known to occur. Allard wonders what efficient barrier may guard the embryo. When we suppose the cause to be a parasite, be it an ultramicroscopic one, it is easy to understand that the embryo is protected from infection. The phloem strands connect the mother plant with the young tuber, but between the plant and the embryo this connection is interrupted twice, once between the mother plant and the endosperm and the second time between the endosperm and the embryo. The embryo feeds by means of osmosis and can only absorb fluid matter. So there is every reason to agree with Allard (1915) that the pathogenic agent is a parasite. That bacteriology has not succeeded in cultivating the organism is no proof that there is no organism present.

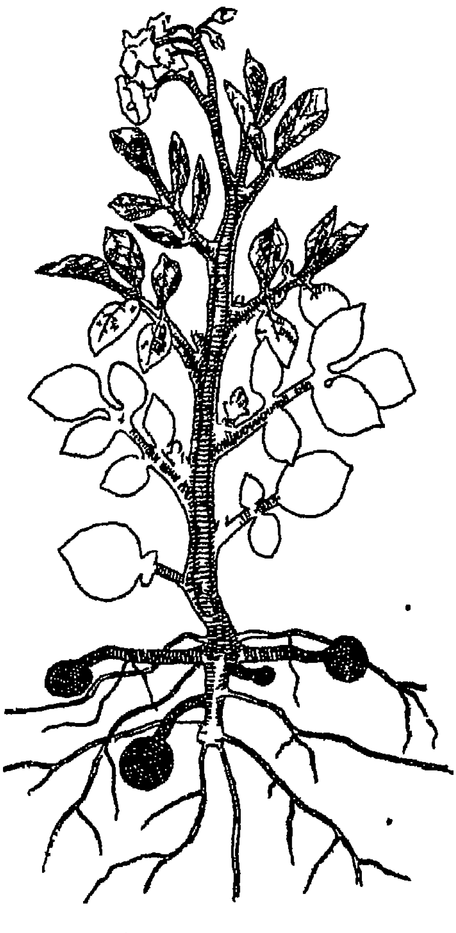

FIG 14 THE SAME EXPERIMENT IN A PLANT WITH PRIMARY LEAF-ROLL

The necrosis of the sieve tubes starts in the top and here only is the downflow of carbohydrates restricted. The stalk, because of this delay, has not been able to convey all its starch to the tubers.

The well known hypothesis of senility of potato varieties as a consequence of vegetative reproduction is explained by the fact that the two diseases which have been considered as degeneration are always propagated by the tubers and exceptionally by sexual reproduction. This hypothesis has been supported also by the fact that plants attacked by phloem-necrosis or mosaic are more susceptible to *Phytophthora infestans* than healthy potato plants of the same variety. There may be yet another reason for the degeneration theory, as the possibility must be admitted that the contagia which cause phloem-necrosis and mosaic will gradually adapt themselves to varieties which at first were highly resistant.

True leaf-roll or phloem-necrosis seems to be prevalent in all countries where the culture of potatoes has some importance. Until recently no data being at hand about its occurrence in France, the author asked Foëx (Paris) to inquire into the matter, and was soon informed (1914) that it prevailed severely in that country. V. D. Goot (Buitenzorg) sent some samples with typical phloem-necrosis from Java, where potato culture is practiced on the highlands. Since its relation with the tobacco mosaic has been shown, the potato mosaic also must be widely spread.

Oortwyn Botjes has shown how from susceptible varieties disease-free (not immune) strains can be isolated; the same method has been applied in getting rid of mosaic. By means of selection, satisfactory results have been obtained in the province of Frisia in maintaining the well known Frisian Eigenheimer, which is susceptible to mosaic. This selection is greatly promoted by the well organized field inspection service in that province. At the commencement of this organization (about 1903) only a very crude selection was made by removing diseased "hills," but gradually some farmers have systematically selected the best plants and from each of them they bred the progeny apart, selecting from it anew the best plant, and so on. Some farmers have had the good fortune to avoid the sources of infection without knowing mosaic to be contagious. But now that they have been informed about the mode of dissemination of this trouble they are more able to avoid reinfection.

One of the most important points in the breeding of new varieties from the true seed must be to pay close attention to their resistance to mosaic and phloem-necrosis. The variation in the asexual reproduction is much more limited than in the sexual one; whether it is possible to obtain resistant strains exclusively by selection is doubtful. Only when sports (mutations) occur, it seems that deviations in susceptibility may be connected with differences in shape and color of the tubers and growth habit of the "vine." This part of the problem is still in its infancy.

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# RAISING PHLOEM-NECROSIS AND MOSAIC FREE POTATOES, AND A SOURCE OF INFECTION WHOSE NATURE HAS NOT YET BEEN ELUCIDATED

J Oortwyn Botjes

Phloem-necrosis and mosaic in potatoes will pass from diseased plants to neighboring healthy ones. In the majority of cases the infection is not observable during the period of growth, but becomes apparent in the progeny, the more so as the distance between the healthy plant and the diseased one has been smaller. On sandy soil that was rich in humus, infection has taken place at 2 meters distance, and it is probable that under certain circumstances it may exceed this limit.

The above method of infection is the only one that has been demonstrated with absolute certainty to play a great part. It is true that after mild winters the disease has been detected in healthy potatoes that had been grown immediately following a diseased crop, but in this case the infection may have proceeded from volunteers or from growing tubers remaining alive in the soil and forming only secondary tubers.

In my own experiments, where one or two years intervened between the two crops, there never was any influence from a preceding diseased crop to be detected, so that it may not be of great practical importance, though its possibility must be taken into account.

Still, while excluding every possibility of infection through neighboring plants or prior cultures, infections of the phloem-necrosis and mosaic were pointed out in my fields in the year 1917. The appearance of these momentary infections can not be explained, but perhaps they must be accounted for by the influence of diseased plants growing at 20 meters distance if not more. Quanjer found a close relation to exist between potato and tobacco mosaic. Since this last disease is spread by aphids, it is possible that these or other insects act as disease carriers in the cases in which spreading has occurred. It is not impossible that plant lice are responsible for the greater spreading power these diseases seem to have on Long Island than in Maine. Therefore, in raising disease-free seed potatoes one should bear in mind that diseased plants are sure to infect their sound neighbors and that infection may be due to a diseased preceding crop, or the presence of diseased plants at a considerable distance.

In the raising of seed potatoes one must begin with healthy plants, which is difficult because in most cases it is not possible to detect primary infection, though secondary infection may be easily found out. Therefore, in selecting the plants reserved for seed, one should not only reckon with their outward appearance, but also with the relative possibility of their being infected. Such plants are to be selected as grow at more than 2 meters distance from diseased ones and in a field where the diseases if present are not severe. Not all the selected plants are likely to prove healthy. Some are sure to produce sick progeny that will infect the others if growing too near. Therefore the tubers of each selected plant should be planted in a row and the rows should be arranged so as to leave a space of at the least 3 or 4 meters distance between them. The space between the potatoes in each row may be 50 cm. if they proceed from the same plant; if not, a space of 3 to 4 meters should be kept. Intervening spaces may be planted with beans, beets, cabbage, or other low growth. The crop should be closely watched throughout the year, and every irregularity should be noted on a ground plan. If some of the progeny of one plant should produce sick plants, all the others proceeding from the same plant should be rejected. It is advisable to remove the sick plants as soon as possible, in order to preclude "infection at a great distance."

For culture only such potatoes are used as proceed from plants that have produced an entirely sound progeny. Some of them are set aside to be planted next year on a nursery plot in the above mentioned way, whereas the others are planted on a reserved field for the increase of planting material.

Nursery plots, as well as reserved fields, should be kept at as great a distance as possible from other potato fields; they must not be manured with stable dung, which might contain potato rubbish, and must have been free from potato culture during the last two or three years.

When hoeing the fields or when lifting the potatoes, care should be taken that no earth is transported from sick plants to healthy ones. The above method of raising disease-free seed potatoes may be applied both in case of phloem-necrosis and of mosaic. With both diseases the infection occurs in the same way.

OOSTWOLD, OLDAMBT
HOLLAND

## PHYTOPATHOLOGICAL NOTES

*Phytophthora cactorum (Leb. et Cohn.) Schroeter.* This interesting fungus was found in September, 1919, attacking the fruits of a pear tree at Kentville, Nova Scotia.

H. T. Gussow.

*Personals.* Dr. Carl Hartley, formerly pathologist in the Office of Forest Pathology Investigations, Bureau of Plant Industry, has resigned to take a position in the Institute for Plant Diseases at Buitenzorg, Java. Dr. R. D. Rands, formerly of the Office of Cereal Investigations of the same Bureau, who went to the Institute at Buitenzorg in 1918, has been engaged there in the study of the brown bast disease of rubber. Two Swiss botanists, Doctor Gaumann and Doctor Paravicini, passed through the United States some months ago on their way to accept positions in the same institution.

Dr. W. H. Rankin, for five years assistant professor of plant pathology in the New York State College of Agriculture at Cornell University, has been appointed officer in charge, Field Laboratory of Plant Pathology, St. Catherines, Ontario, in the Canadian Department of Agriculture, and has entered upon his duties.

Mr. Emery D. Eddy, who has been engaged during the past year in the investigation of diseases of market vegetables, for the Department of Agriculture, recently resigned his position to take up commercial work.

# *ABSTRACTS OF PAPERS PRESENTED AT THE ELEVENTH ANNUAL MEETING OF THE AMERICAN PHYTOPATHOLOGICAL SOCIETY, ST. LOUIS, DECEMBER 30, 1919, TO JANUARY 1, 1920.*

*Recent studies on Septoria of wheat.* RICHARD O. CROMWELL

Septoria of wheat in the imperfect stage survives the period from harvest time until the rosette stage of winter wheat Pycnospores retained vitality on leaves of the 1919 crop until early in October. Mature pycnidia were found on volunteer and winter wheat seedlings as early as October 2 and November 1, respectively. Spores were viable on November 10, the date of last test. Artificial infection was secured with spores from the 1919 crop and later from volunteer plants. The maximum and optimum temperatures for germination of pycnospores are 34, and 24 to 28°C., respectively. The time for germination varies between twenty-five and twenty-four hours, depending upon the stage of maturity and certain other factors.

Septoria was most readily isolated by using material which had a large percentage of the spores in condition for rapid germination and growth. On potato agar the young growth is disciform, mucose, and pink to lilac; later it is restricted, mucose to waxy, and pink to dark gray. During the first thirty days mycelia are meager, and spore like (pionnotes like) bodies abundant. These bodies, resembling pycnospores in size, form, and septation, function in producing artificial infection. Old cultures are composed entirely of short, swollen, chlamydospore-like cells. On sterilized wheat straw pycnidia become visible in eight days.

*An epidemic of Fusarium blight (scab) of wheat and other cereals.* A. G. JOHNSON, JAMES G. DICKSON, and HELEN JOHANN

An unusually severe epidemic of Fusarium blight or scab occurred on both winter and spring wheat during the past season Rye, oats, and barley were also attacked. The disease was particularly severe in the Mississippi Valley and eastward. The winter wheat throughout this area was universally attacked, infection ranging from a trace to fifty per cent. The spring wheat, particularly where planted in the winter-wheat area, was infected unusually heavily. In numerous cases large fields of Marquis wheat were left unharvested because so badly blighted.

An extensive survey over fifteen states, correlated with an intensive survey over nine states in the winter-wheat area, brought out some pertinent facts with reference to the epidemic. A careful study of over one thousand specimens collected in the fifteen states showed *Gibberella saubinetii* (Mont.) Sacc. to be the chief causal organism. Less than 1 per cent of the specimens yielded *Fusarium culmorum* (W. Sm.) Sacc., *F. avenaceum* (Fr ) Sacc , and other Fusarium species. The highest infections occurred where wheat followed corn or wheat in the rotations, or where old grass refuse had been allowed to accumulate in fence rows and field margins. The abundant presence of the parasites and the moist, warm weather following the blossoming period of the grain favored development of the disease. Detailed investigational work is in progress on various phases of the problem, including control measures.

*The effect of temperature and light on Fusarium sp. causing wheat scab.* JEAN MACINNES

An investigation was made of the effect of temperature and light on the species of Fusarium commonly causing wheat scab in Minnesota. Repeated trials show that the minimum temperature for growth is approximately 5°C., the optimum near 25°C., and the maximum between 30 and 35°C. Field experiments showed that a greater percentage of wheat heads became scabby when the temperature at the time of their emergence from the sheath was about 76°F. (24.4°C.) than when the temperature was lower. Apparently high humidity is conducive to the development of the disease.

Both diffuse light and sunlight injured the viability of spores. Only about 1 per cent of the spores from scabby heads which were exposed all winter to light either outside or indoors germinated in the spring, while the spores from heads which had been kept in the dark germinated readily. The average percentage of germination of spores kept in the dark outside was nearly twice as great as that of those kept in the dark inside.

*Foot-rot of wheat and its fungous associates.* F. L. STEVENS

Foot-rot of wheat as found in Madison County, Illinois, is described in general and histologically and the more significant fungi associated with this disease are discussed.

*The resistance shown by three hard winter wheats, Kanred (P762), P1066, and P1068 to plant diseases.* L. E. MELCHERS

Various publications by J. H. Parker and the writer have called attention to the resistance of these wheats, products of the Kansas Experiment Station, to stem rust (*Puccinia graminis tritici*). Their use as differential hosts for physiological testing of rust strains is quite outstanding. Unpublished data conclusively show that they are resistant to leaf rust in Kansas. No statements are ventured as to whether the leaf-rust infection during six years observations was caused by one or more biologic strains. The field behavior of Kanred throughout Kansas the past season to the leaf and stem-rust epidemic was striking. This is especially interesting because of the great commercial value of Kanred.

C. E. Owens, of Oregon, states that in an induced epidemic of stripe rust, 1919, these varieties remained free of rust at all times. Their resistance to stripe rust in the field and greenhouse, however, has not been constant. E. C. Stakman has stated that all three varieties are only slightly susceptible to *Puccinia graminis tritici-compacti*. This marked resistance is not maintained, however, for all biologic forms of stem rust. F. D. Heald has unpublished data which show that Kanred shows resistance to stinking smut of wheat when compared with other commercial varieties. Kanred is also showing resistance to other diseases in varietal tests, but specific mention of these is being withheld for the present.

*The resistance of Kanred (P762), P1066, and P1068, three hard winter wheats, to leaf rust.* LEO E. MELCHERS AND JOHN H. PARKER

Careful observations the past six years have shown that these varieties are markedly resistant to leaf rust under Kansas conditions. The average percentages of leaf rust for these seasons were consistently low, 9.5 per cent in the case of Kanred (C. I. 5146), 6.2 per cent for P1066 (C. I. 5879), and 5.7 per cent for P1068 (C. I. 5880). Leaf rust as it occurs in Kansas, however, may or may not show the same biologic specialization as the leaf rust occurring in other regions.

A severe epidemic occurred in the state the past season, some wheat fields being plowed up because they were so severely injured. Approximately 50,000 acres of Kanred were grown in 1919. About eight hundred fields, belonging to 572 growers, were inspected, these fields being located in forty-six counties in different parts of the state. The percentages of leaf-rust infection in all these Kanred fields were strikingly uniform, ranging from 8 to 10 per cent. Adjoining fields of Turkey and Kharkov, commonly grown varieties, showed from 20 to 100 per cent leaf rust, the majority approaching the latter figure. The varieties P1066 and P1068 are not being grown commercially, but showed even more resistance than Kanred at the Experiment Station.

*The development of loose smut of wheat as modified by soil fertility.* F. D. Fromme.

In a series of field plots subjected to continuous wheat cropping with varying applications of fertilizers, the percentage of heads affected with loose smut (*Ustilago tritici*) varied according to the degree of soil fertility maintained. The highest percentages of smut are found in plots of low fertility, and vice versa. Similar observations were made in connection with stinking smut (*Tilletia laevis*) in greenhouse plots. It seems probable that these variations are due to the total or partial elimination of the smut fungus by the greater vigor of the growth of the plants on the more fertile soils.

*Relation between soil moisture and bunt infection in wheat.* Chas. W. Hungerford and A. E. Wade

Field observations during the last four years in Lewis County, Idaho, which is in the area where soil infestation by bunt is common, have shown a close relation between the amount of bunt on winter wheat and the amount of moisture in the soil at the time of planting. Observations have been made in a number of fields of wheat where seed from the same lot was sown before and after a rain. Only a small amount of bunt was found in the portions of the fields sown in rather dry soil, which contained, in most cases, close to the minimum amount of moisture necessary for good germination of the seed. On those fields those portions sown after it had rained contained in some cases over 30 per cent of bunt.

During the past summer the following experiment was carried on both on the Experimental Farm of the Agricultural College at Moscow, Idaho, and at Nezperce, in Lewis County, Idaho. Six large soil containers were filled with soil containing different amounts of moisture varying from saturation to less than enough to cause good germination of the seed. The soil was heavily smutted and the seed was covered with viable spores of *Tilletia tritici.*

The following table gives the results of the tests:

| *At Nezperce, Idaho* | | *At Moscow, Idaho* | |
|---|---|---|---|
| *Soil Moisture* | *Smut per cent* | *Soil Moisture* | *Smut per cent* |
| 7.7 | 0 | 7 | No growth |
| 15 | 7 | 12 | 0 |
| 20 | 26 | 17 | 25 |
| 24 | 27 | 21 | 30 |
| 36 | 62 | 25 | 68 |
| 38 | No growth | 38 | 100 |

This work is being repeated in both greenhouse and field and under more accurately controlled conditions.

*Experiments on the control of stinking smut of wheat.* G. H. COONS

Samples of wheat of seventeen varieties, heavily dusted with *Tilletia laevis* and *Tilletia tritici*, respectively, in September, 1917, were planted in 1918, and gave in 1919 yields free from any trace of *Tilletia tritici*, but with abundant *Tilletia laevis* infection. Striking differences in the amount of smut due to *Tilletia laevis* were evident in the series, certain wheats, such as Little Club and Zimmerman, showing 60 per cent or more of smut, while Turkey P889, Turkey P1038, Nebraska Hybrid, and Fultz showed 4, 4, 2, and 0 per cent respectively. In many cases the stand was sharply reduced in the smutted series, the reduction being greatest in those varieties showing most smut.

Best smut control was obtained with heavily smutted Red Rock wheat by the "soak and skim" method, using 1 pint of formaldehyde to 40 gallons of water, no trace of smut appearing in the plots. In sprinkling experiments, using the same solution, if the grain was sprinkled until wet and covered four hours, control was perfect. With less time of covering or with only 1 quart of solution to the bushel control of smut was poor.

The "dry" treatment, using 1 pint of formaldehyde plus 1 pint of water to 50 bushels of grain, gave perfect control. Lesser amounts of formaldehyde and greater dilution with water gave poorer smut control.

Slight injury to stand seems incident to any formaldehyde treatment, but unless covering is long or strength of solution excessive this effect is compensated by the growth of stalks. The utilization of a follow-up spray or dip of ammonia to neutralize the formaldehyde by the formation of Hexamethylene tetramine is suggested.

*Evidence of disease resistance in barley to attacks of Rhynchosporium.* A. G. JOHNSON and W. W. MACKIE

The disease of barley and rye caused by *Rhynchosporium secalis* (Heinsen) Davis has been noted in limited amounts in the upper Mississippi Valley for a number of years. In this section the attacks have been, on the whole, rather scattered and only occasionally severe. In certain sections on the Pacific coast, especially in California, this disease has been more severe, particularly on barley. Differences in susceptibility of various barley varieties have been previously noted by others. During the summer of 1919 the writers, in examining a large series of barley varieties growing at Davis, California, noted what seemed to be evidence of disease resistance in only two varieties in the entire series, all others being heavily attacked. These were three different selections of Tennessee Winter and one selection of Mariout. Other selections from these same varieties grown in adjacent rows in the same series of plots were heavily attacked, as were all the other varieties.

*The purple sheath spot of corn.* L. W. DURRELL

Corn is universally affected by a disease producing purple blotches and spots on the leaf sheaths. This was early attributed by Burrill to bacteria, which in the first part of the season also caused a dwarfing of certain plants in the field. The spots are external manifestations of necrosis of the tissues on the inside of the sheath. They appear only after the fall of the pollen and late in the season are present on practically every corn stalk.

Over the diseased areas within the sheaths are to be found great numbers of dead pollen grains and pollen sacs that have worked past the ligule and into the moist enclosure of the sheath. These pollen grains and sacs act as media for saprophytic organisms, which later enter the sheath tissues. Sugary secretions of aphis may act in a similar way. The exclusion of pollen and aphids from the leaf sheaths has been found to eliminate the spot. On the other hand the introduction of pollen or pollen sacs exposed to spores of various molds and other fungi produces the spot. The introduction of mycelium or spores of these fungi however has no effect unless accompanied by a small bit of agar. Sections of sheath spots show stomatal invasion by fungi and bacteria, prior to necrosis of the tissues.

*Some results of corn root rot work in Ohio.* W. G. Stover

In coöperation with farmers in several counties, seed corn was tested for germination, each lot being divided into several groups and planted on the farms. In September no difference could be seen between the various groups except in one instance. In this case the group planted from ears which showed Fusarium-like fungi in the germinator was much poorer than the others. In several cases an application of manure or nitrate of soda resulted in a great improvement in the appearance of the corn in all the groups.

Two hundred and eleven ears were tested a second time and planted in separate rows. The average yield of the rows planted from ears free of Fusarium-like fungi exceeded the others at the rate of more than 4 bushels per acre. Data were also collected on the relative numbers of ears, nubbins, and barren stalks in these rows. There was a marked variation in the results of the two germination tests.

*Corn root and stalk rots.* J. R. Holbert and G. N. Hoffer

Corn generally is more or less affected by root and stalk rots. In the principal corn growing sections the losses resulting from these diseases range from traces to 25 per cent and more. The symptoms so closely resemble and are so intimately related to the effects of drought, low fertility of soil, poor drainage, insect and frost injuries that their diagnosis is sometimes difficult. Probably the greatest loss due to these rots is caused by using infected seed. Many kernels from badly infected seed ears either do not germinate or produce seedlings that die before reaching the surface of the soil or soon afterwards. Other infected kernels produce chlorotic and blighted seedlings and stunted stalks. This early retardation of growth of infected seedlings is responsible for much barrenness and many nubbins. Badly infected kernels and seedlings may cause a secondary rotting of the roots of other seedlings and plants in the same hill. Corn that is planted in infected soil may become infected later in the season. These secondary infections also cause a retarded growth and subsequent barrenness and nubbin production. Other symptoms resulting from secondary infections are leaning and down stalks, broken stalks, broken shanks, wilted plants, and prematurely dead plants. Secondary infections may cause barrenness, due to the abortion of the reproductive parts. Ears borne on infected plants are usually more starchy in composition than ears borne on healthy plants. Such ears are unsatisfactory for seed purposes.

The pathogenes are chiefly species of Fusarium and Gibberella. The common wheat scab organism, *Gibberella saubinetii* (Mont.) Sacc., is probably the most common pathogene responsible for much of the root and stalk rotting of corn plants in the Central States.

The most successful control measure that has been developed so far is the selection of seed from healthy, vigorous plants that show no evidence of disease upon germination, culling out by physical selection those ear types that have been found to be more susceptible to these troubles. Breeding experiments are being conducted to develop resistant strains and varieties.

*Iron accumulation and mobility in diseased cornstalks.* G. N. HOFFER AND R. H. CARR

The internal tissues of each node in a corn stalk can be differentiated into two zones, the upper, Zone A, functioning in starch accumulation and the growth of the internodes, and the lower, Zone B, where the fibrovascular bundles branch to form the bundles extending into the roots, leaves, and husks of the ears.

Iron and other metallic bases accumulate in organic combinations in the Zone B tissues, first in the phloem cells, and later in the walls and lumina of the zylem elements. The central bundles of the lowest nodal tissues in the stalks are the first to accumulate iron compounds in greatest quantities. The amounts which accumulate seem to be directly related to the premature dying of the lowest leaves. This phenomenon is commonly referred to as "firing."[1]

When the corn stalk show the effects of malnutrition and the symptoms of root rot infections, the Zone B tissues are frequently brown to brownish-purple discolored. Iron compounds are invariably present in larger quantities, as determined by chemical analyses, and oxidase and catalase activities are greater in the nodes of infected stalks than in those of normal healthy stalks of the same age in the same soil. Aluminum and manganese compounds have also been found in greater quantities.

The H-ion concentration in the Zone B tissues approaches neutrality (pH = 6.2-6.8) in the stalks showing the disease symptoms of root rots, whereas in the normal stalks the concentrations approximate those of the internodal tissues (pH = 4.0-5.2).

By testing stalks showing various symptoms of malnutrition and infection by root- and stalk-rot organisms, it was found that water, colored slightly with methylene blue, was not conducted in the vessels in the nodes which showed the largest accumulations of iron in them. Healthy stalks would always have practically all of the vessels functioning. Cross sections of the bundles in the Zone B tissues of healthy and diseased stalks show varying quantities of iron compound deposits in them, the greater quantities appearing in the bundles of diseased stalks.

It is believed that the metallic bases, with special reference to iron, are responsible for causing changes in the normal functioning of the Zone B tissues, first, in the translocation of carbohydrates from the leaves to the stem and roots by the phloem elements, and later in the conduction of water and mineral nutrients in the transpiration current.

If iron compounds are present in seedlings in supra-optimal quantities they will be noticed first in the lowest Zone B tissues, which are located at the distal end of the mesocotyl. When this accumulation occurs very early in the life of the young plant it is associated with a retarded growth of the plant.

Corn plants with the upper leaves chlorotic are characterized by low H-ion concentrations and contain immobile iron compounds within them. The Zone B tissues at the bases of the chlorotic leaves give no reaction for the presence of iron, whereas in the lower tissues iron compounds can be found. No tendency toward chlorosis was found in any of the stalks which had a relatively uniform H-ion concentration of pH = 4.0-5.0 in the Zone B tissues. The rates of growth of young plants and the degree of chlorosis may be determined by the relative abilities of the plants to develop and maintain a sufficiently high H-ion concentration to keep the iron compounds mobile as occurs in normal green plants.

*II. Experiments to test effects of iron salts on corn plants.* G. N. Hoffer and R. H. Carr

In order to test the capacity of the Zone B tissues of healthy corn stalks in the field to accumulate iron and other metallic base compounds, 0.5 per cent, 1 per cent, and 2 per cent solutions of iron sulphate, iron chloride, iron citrate, iron carbonate, iron nitrate, dialyzed iron, copper sulphate, aluminum chloride, and aluminum sulphate were used. Punctures were made in the internodal cortical tissues just above the upper nodes bearing roots. The solution were introduced through glass tubes inserted in the stalks.

The ferrous sulphate solutions were very active in affecting the Zone B nodal tissues. Catalase and oxidase actions were greatly increased. The tissues became brown and disintegrated, the leaves wilted and fired, and premature death of the stalks resulted. The nodal tissues were heavily laden with iron compounds and presented an appearance similar to those of infected stalks. Approximately 25 cc. of a 0.5 per cent solution introduced into healthy stalks 6 to 8 feet high proved effective. The effects of the treatments were similar to those observed in stalks affected by root rots.

The ferric salts of equal concentrations and quantities were either less effective or had no apparent effects whatever. Ferric citrate, however, in a 2 per cent solution darkened the bundles and caused a wilting of the leaves. In all cases the ferric salts stimulated oxidase and catalase action, but not to the same degree as the ferrous sulphate solutions. Potassium sulphate in a 2 per cent solution had no noticeable effect in the same period of time. Dialyzed iron was not appreciably absorbed.

Aluminum salts in equivalent concentrations also stimulated catalase and oxidase action in the Zone B tissues. Copper sulphate in a hundredth normal solution was very toxic, both the Zone A and Zone B tissues of the nodes were destroyed. The copper compounds could be detected in quantities in both sets of tissues.

Check plants supplied with water or with nutritive salt solution in equivalent concentrations showed no harmful effects from the treatment.

*Correlation of early growth, variation and productivity of maize as influenced by certain pathologic factors.* J. R. Holbert, James G. Dickson, and H. Howard Biggar

Inoculation experiments were conducted during the season with Funk's Ninety Day corn selected from apparently healthy plants and diseased plants. The ears differed in their disease reading on the germinator. They were used in ear-to-row plots on soil that had not grown corn for the previous twelve years. In each row alternate hills were inoculated at planting time with *Gibberella saubinetii* (Mont.) Sacc. from wheat. In the inoculated hills germination was lowered, early growth retarded, relative vigor through the season reduced, storm resistance decreased, and average grain production lessened. The plants in the uninoculated hills planted with apparently disease-free seed from healthy plants remained normal. *G. saubinetii* was readily recovered from the diseased plants in the inoculated hills, especially during the seedling period.

Plants, strong or weak in the early stages of growth, had a tendency to retain this relative vigor throughout the season. Average grain production was directly correlated with rate of early growth and early vigor. Seed selected from prematurely ripe plants, but showing no evidence of disease on the germinator, lacked the ability to compete successfully with the disease-free seed selected from plants maturing normally. Apparently disease-free germinating ears selected from healthy, vigorous plants produced plants that had a more rapid early growth, more storm resistance throughout the season and a higher average grain production than diseased germinating ears similarly selected

*Apple blister canker* W O GLOYER

The following conclusions have been reached after seven years study of the blister canker (1) The canker caused by *Nummularia discreta* Tul is not as important economically in New York as in the middle west (2) The fungus is more actively parasitic on Sorbus than on Malus (3) Forms varying in pathogenicity and cultural characters sufficiently to be called varieties or strains are produced by varying the substratum upon which the parasite grows (4) It may be active in the wood and yet show but little canker formation (5) The presence of the fungus in the wood is one cause of slime flux (6) Cankers may enlarge at any time between early spring and late autumn They enlarge slowly during periods of drought and become more active after rains following a drought (7) Shellac followed by coal tar was found to be the most satisfactory covering for pruning wounds (8) The covering is not the controlling factor in the healing of a cankered wound (9) The physiological condition of the host determines the activity of the parasite (10) Parasitism is complex and can not be measured in terms of percentage of water content of the wood as indicated by Munch

*A preliminary report on apple scab and its control in Wisconsin* G W KEITT

During the past season, the discharge of ascospores of *Venturia inaequalis* (Cke ) Wint was carefully followed at Madison and at Sturgeon Bay At Madison it began on April 20 and ended about June 7 The heaviest discharges occurred prior to May 8 The blossom buds did not separate in the clusters until about May 11-18 the time varying with varieties

At Sturgeon Bay the first ascospore discharge was noted on May 15 and the last on June 16 The heaviest discharges occurred prior to May 25 The blossom buds did not separate in the clusters until about May 26 30

At Sturgeon Bay on certain badly scabbing varieties lime sulphur and Bordeaux mixture failed to control scab satisfactorily when applied comparatively according to the standard four spray schedule (a) May 26 27 as soon as the blossom buds separated before blooming (b) June 4 5, when three fourths of the petals were off (c) June 18 19 and (d) August 20 The addition to this schedule of an application on May 16 17 soon after the blossom buds became exposed in the clusters, led to excellent control

*Results of spraying the apple for blotch in Ohio in 1919* W G STOVER, F H BEACH, AND T H PARKS

Spraying Clermont County orchards in 1918 at three weeks intervals following petal fall gave less than 30 per cent marketable fruit Three applications of Bordeaux mixture in 1919 in several counties at intervals of two weeks followed by an additional application in July gave an average of over 90 per cent marketable fruit Unsprayed trees in the same orchards gave less than 10 per cent marketable fruit in the blotch areas Sprayed trees bore more fruit and their apples were larger as a rule than those from unsprayed trees No trees were sprayed on the three-weeks schedule in these trials

*A stem disease of tomato caused by Macrosporium solani E. & M.* JOSEPH ROSENBAUM

A stem disease of tomato plants was found doing considerable damage the past season in Delaware. The disease has been found on all ages of plants. Affected stems, at the point where they emerge from the surface of the soil, are dark brown, shrivelling up with age, and a girdling effect is produced.

Numerous isolations and inoculations establish the fact that a fungus belonging to the genus Macrosporium is the causal organism.

Comparison of this Macrosporium were made with *M. solani* from potatoes and with Macrosporium cultures isolated from typical "nail head" spots on Florida tomatoes. These comparisons show that the Macrosporium discussed here is different from the one isolated from the "nail head" spots from Florida, in that the former is unable to produce infection on uninjured green fruit, as well as differing in culture and morphology. It resembles *M. solani* from potatoes in the kind of lesions produced when used for inoculation on fruit and stems, and in morphological characters. It should therefore be referred to as *Macrosporium solani* E. & M.

*Field tests with strains of wilt-resistant tomato seed in 1919.* J. A. MCCLINTOCK

Strains of seed from wilt resistant tomatoes from various sources were tested on a heavily infested piece of soil at the Georgia Experiment Station in 1919 and no strain proved sufficiently resistant to grow throughout the summer.

Strains of commercial peppers and eggplants grew and fruited throughout the summer on the same infested soil.

*The Alternaria fruit rot and Rhizoctonia stem rot of tomatoes.* MEL. T. COOK

This rot appears in three forms: (a) following the infection of cracked fruit, (b) following the infection of sun burned fruit, (c) nail head spot on green fruit. This papers refers especially to the second. Most of the sun burn is due to a loss of foliage following attacks of *Septoria lycopersici*. The fruit is most susceptible to sun burn just as it begins to color. The exposed surface becomes yellowish instead of red. This yellowish area begins to freckle with brown spots which enlarge, become black, rupture and show masses of spores. Cultures from youngest spots gave no growth but as the spots increase in age and size, it becomes easier to get growths.

The stem rot which was very prevalent in South Jersey in 1919 was due to Rhizoctonia attacking the stems at the surface of the ground. The disease evidently started in the seed bed but continued in the field causing heavy losses. This same condition was found in one field of cabbages.

*Spacing tomato plants for field spraying.* C. E. TEMPLE

It has been found from experiments extending over four years that the rows of tomato plants must be set approximately 6 feet apart in order to spray with large machinery without great loss from mechanical injury to the plants. On the other hand, the plants may be set close enough in the row to obtain the usual number per acre without loss from over-crowding. In Maryland the most desirable spacing for spraying purposes is three feet by six feet. Other advantages from this method of setting are: That cultivation may be continued later in the season than usual to conserve moisture, that a cover crop may be planted at any time without injury to the plants, that the plants interlock in the row so that they are not tossed about by the wind as much as when checked four or five feet each way, and that the crop may be harvested and hauled from the field without trampling the vines.

*Tomato spraying experiments for the control of Septoria.* C. E. TEMPLE

During the past four years an attempt has been made to devise a practical and effective method for controlling the Septoria leaf spot of tomatoes under field conditions. More than 200 acres of tomatoes were divided into plots and used in these tests. As a rule every third plot remained untreated as a check. On the other plots both liquid and dust sprays were used and these were applied in almost every case by traction or gasoline engine machines. A large number of spray formulas were tested, but the bluestone liquid sprays, both with and without rosin oil soap, were the most promising. These sprays were run in series to determine the most profitable number of applications to apply and also to determine the quantity of spray material to use. The formulas were applied at the rate of 50 gallons and 100 gallons per acre to determine the most economical quantity needed for the control of the disease. The results were variable, ranging from no increase in production in a few cases to more than 100 per cent increase in others. There was, however, usually a substantial increase over the checks wherever at least four applications of a Bordeaux or a Bordeaux soap were applied.

*Effect of straw mulch on potato leaf roll.* W. O. GLOYER

In 1918 plots of Sir Walter Raleigh potato on clay loam were covered with 3 inches of short straw while others were cultivated. When in bloom the leaf-roll plants were rogued and 267 hills in the mulch and 511 hills in the cultivated plots showed 11.6 and 11.4 per cent respectively. On the mulch plots the plants were of a yellower tinge, were killed earlier by frost, and had larger and flatter tubers than did the cultivated plants. The yield per plant averaged 630 grams in the former and 603 grams in the latter.

In 1919 the highest yielding hills from these plots were planted as units and given normal cultivation. The 197 hills from the mulch and 78 hills from the cultivated plots showed 59 and 28 per cent of leaf roll, respectively. In some units there was a tendency of the smaller tubers to produce disease-free plants. Conclusions: (1) Mulching did not overcome the infectious nature of the disease; (2) in badly diseased fields even tubers of the healthy-appearing and high-yielding plants are unsafe for seed.

*Sulfur experiments for the control of potato scab.* WILLIAM H. MARTIN

Sulfur was broadcasted at the rate of 300, 400 and 600 pounds per acre just before planting. The 300 pound applications gave approximately as good results as the larger amounts. There was a marked increase in salable primes from the sulfur treatments.

Soil samples were taken at the start of the experiment and again when the crop was dug and the hydrogen ion concentration of the soil extracts determined colorimetrically. The samples examined before the sulfur applications were made were found to be slightly acid. An examination of the soil extracts from the different plots at the close of the experiment showed marked changes in hydrogen ion concentration. The untreated plots remained unchanged while there was an increase in acidity (decrease in exponent) where sulfur was applied at the rate of 300, 400 and 600 pounds per acre, the mean for the plots being 4.9, 4.9 and 4.5 respectively.

*The 1919 survey for potato wart.* G. R. LYMAN

During the past season the Plant Disease Survey, in cooperation with the Federal Horticultural Board, conducted an extensive survey in an effort to determine the geographical distribution in the United States of potato wart, which was discovered in the autumn of 1918 in a large area in the neighborhood of Hazleton, Pa. Immense quantities of European potatoes, some of which may have been affected with wart, were imported before the imposition of the Federal Quarantine in 1912 and were distributed very widely. Hence the greater portion of the United States was under suspicion of harboring the disease, especially home gardens in cities and in other districts which are not in close touch with modern agricultural methods and workers. On account of the immense area to be covered in a short time with limited funds, the Plant Disease Survey endeavored to enlist the interest and assistance of as many helpers as possible. This effort was conspicuously successful and the survey was marked by the hearty and effective cooperation not only of the Survey's collaborators, but of growers and users of potatoes generally. Wart was found in small amounts in six villages in western Pennsylvania and in two villages in northern West Virginia, all being in the soft-coal section, except one of the West Virginia localities. Apparently no other extensive area of infestation exists at all comparable to the Hazleton district, but undoubtedly there are other undiscovered isolated points of infestation in regions out of touch with modern agriculturists. These can be brought to light only by further intensive survey.

*Wood-destroying fungi in pulp and paper mill roofs.* R. J. BLAIR

When the roof of a paper mill is built of wood moisture in the air permits the development of wood-destroying fungi in the timber. Rapid rotting takes place and it is often necessary to renew the timber after six or eight years' service. The roofs of eighty of the Canadian paper mills have been examined to find the different types of construction used and the services secured from each. The common faults in every case are that moisture enters the spaces between the planks and reaches their upper side, where it condenses, causing a moist condition of the planks during prolonged periods and at the same time the wood is of a non-durable species. There is no evidence that other species of fungi than three of those mentioned last year in connection with the cotton mill roofs, *Lentodium tigrinum*, *Lenzites trabeum*, and *Poria xantha*, cause these losses.

The remedy for such timber decay lies in a combination of four factors, which need consideration when the roof is being built. These are the use of wood which resists decay, ventilation which carries away the moisture, liberal dry-air heating, and a heat-insulating layer placed on the upper side of the planks.

*Black crust of Brazil nuts.* E R. SPENCER

Brazil nuts are frequently affected by a fungus disease that is not noticeable until the nuts are shelled The kernel is covered by a black mycelium, which destroys the outer cell layers.

On cornmeal agar the fungus grows well but slowly. A greenish black thallus, which produces very few, hairy, globose pycnidia after several weeks results from a direct planting

The conidia are at first hyaline and single celled, but later become two celled and dark, with conspicuous striations. The spores are about 21 by 13 $\mu$.

*Leaf hoppers and hopperburn of potato leaves.* J. E. KOTILA

During the summer of 1919 experiments were performed to ascertain whether or not tip burn of potato leaves was caused by leaf hoppers and to determine if aphids had a similar effect. Data obtained from these experiments are tabulated below.

*Effect of leaf hoppers on potatoes*

| PLANTED | CAGED* | NUMBER OF CAGES | NUMBER OF PLANTS | TREATMENT | DATE | RESULTS | REMARKS |
|---|---|---|---|---|---|---|---|
| Field experiments, Upper Peninsula Experiment Station, Chatham, Michigan | | | | | | | |
| July 2 | July9 | 3 | 6 | 25 Leaf hoppers (Empoasca mali LeB, introduced into each cage | August 1 | Burn | First sign of injury observed August 1. Leaves showed light-colored margins. August 14, margins of leaves brown. Serious burning in all 3 cages |
| July 2 | July 9 | 3 | 6 | Check. No insects | August 1 | No burning | Leaves normal |
| July | July 9 | 1 | 2 | 25 Potato aphids introduced | September 4 | No burning | September 25, leaves green |
| Greenhouse experiments, Michigan Agricultural College, with potted potato plants | | | | | | | |
| September 20† | At once | 1 | 3 | 25 potato leaf hoppers introduced | September 22 | Burn | October 1, leaves showed marginal injury |
| September 20† | At once | 1 | 3 | Check—no insects | September 22 | No burning | October 1, leaves green |

* Cages were set up July 9 before plants broke through soil and were of sufficient size to cover two hills. No insects on plants prior to date leaf hoppers or plant lice were introduced.

† Sprouted tubers were potted on this date.

The results obtained from the above experiments are in accord with those obtained by Dr. E. D. Ball in 1918, report of which was published in Phytopathology, 9: 291-293, July, 1919, and confirm his conclusions that the potato leaf hopper is responsible for the injury to potato leaves known to pathologists as "Tip Burn," but which he more aptly terms "Hopperburn."

### *The relation of soil temperature and soil moisture to the occurrence of cabbage yellows.* W. B. TISDALE.

The results of greenhouse experiments with cabbage seedlings grown in "sick" soil held at constant temperature for twenty days show that yellows does not occur below 17°C. or above 34°, the optimum temperature being 25° to 28°C. This range compares favorably with the growth of the fungus (*Fusarium conglutinans*) in pure culture. Seedlings of the resistant cabbage strain, Wisconsin Hollander, are relatively susceptible until they are at least thirty days old. These results are supported by field trials on "sick" soil at Racine, Wis., during the seasons of 1917 and 1919. Continuous soil temperature records have been secured and correlated with the occurrence of the disease both in the regular field crops and in seedlings from successive plantings made at various intervals. The incubation period varies from one day at optimal temperature to sixteen days at 17°C., the time being reckoned from date the seedlings break through the soil surface.

Experiments were conducted with "sick" soil in which the moisture content was kept constant with Livingston's auto-irrigators and the temperature held at 20 to 22°C. The water holding capacity of the soil was 31.36 per cent figured on wet weight. The yellows occurs and develops most rapidly in soil with 19 per cent moisture, less rapidly with 26 per cent, and still less rapidly with 23 per cent moisture.

### *The influence of soil temperature on the development of potato scab.* L. R. JONES AND H. H. MCKINNEY

Seasonal and regional variations have been observed in the occurrence of common scab on potatoes caused by *Actinomyces scabies* which seem to be correlated with climatic conditions. Opportunity has as yet permitted the investigation of the influence of temperature relations only. Evidence has, however, been secured that soil temperature is so important a factor that it may account for much variation. Greenhouse cultures with soil temperatures of 12°, 15°, 18°, 21°, 24°, 27°, and 30°C. have shown that the optimum for tuber development is about 18° to 21° while that for scab development is about 24°C. At higher temperatures the number of infections continues large but there is inhibition of tuber growth and associated size of lesions. In experimental field plots it has thus far been impossible to hold uniform soil temperatures. We have, however, by the use of mulches and cooling pipes with small plots secured data which is in essential accord with that from the greenhouse cultures.

### *The relation of soil temperature to the development of the tomato Fusarium wilt.* E. E. CLAYTON

As a preliminary to the main work, the growth reactions of the fungus and normal plant was determined for a wide temperature range. Petri dish cultures of *F. lycopersici* were exposed to temperatures ranging from 8° to 38°C. for nine days. The optimum for growth was found to be about 28°C., the minimum 9° to 10°, and the maximum 37°. Very abundant growth, however, was secured at temperatures ranging all the way from 18° to 31°. The optimum soil temperature for the growth of the normal tomato plant was found to lie between 25° and 30°C., the maximum being a little above 35° and the minimum a little below 12°. Tomatoes grown in soil infected with pure cultures of *F. lycopersici* were subjected to soil temperatures ranging from 19° to 35°C., inclusive. The optimum soil temperature for the development of the disease proved to be about 28°C. The disease rarely appeared at temperatures lower than 22° or higher than 32°. These trials were carried on at Madison, Wis., and the time of exposure to the different soil temperatures varied with the separate trials from four to six weeks during the late winter months, February–April.

*Occurrence and control of black leg of cabbage.* J. C. WALKER

Part of a seed bed sown at Madison, Wis., with infected seed and protected from rain showed 0.5 per cent infected plants after three months; that portion exposed to natural rainfall, 29 per cent; that frequently sprinkled, 39 per cent. Seed sown from same lot at La Conner, Wash., where dry weather prevailed, showed no disease after seven weeks. Rainy periods seem essential for dissemination of the fungus from primary centers. Correlation of rainfall and occurrence of disease in various localities indicates that weather conditions during growth of seed bed are extremely important in development of epiphytotics. The fungus is not entirely eradicated from infected seeds treated with 1–500 mercuric chloride for one hour; 1–128 formaldehyde for one hour; water at 55°C. for ten minutes; dry heat at 85°C. for seventy-two hours, 90°C. for forty-eight hours, 95°C. for twelve hours. Thus seed treatment within limits of injury to germination is not completely effective, and field observations show that proper weather conditions may still cause epiphytotics notwithstanding treatment.

*Fifth progress report on Fusarium-resistant cabbage.* L. R. JONES, J. C. WALKER, W. B. TISDALE.

Since last reported (Phytopath. 6: 102; Wis. Agr. Exp. Sta. Res. Bul. 38), the Wisconsin Hollander has maintained its character of relative resistance wherever tested and is now grown almost exclusively on the "cabbage sick" soils of Wisconsin. With the coöperation of a skillful grower (W. J. Hansche) an equally resistant earlier strain of Wisconsin Hollander has been selected. Resistant strains have also been perfected of two leading kraut varieties, All Seasons and Brunswick, and other promising variety selections started. In coöperation with the United States Dept. of Agric., Office of Cotton, Truck and Forage Crop Disease Investigations, and the National Kraut Packers Association, the problems of production and distribution of seed on a commercial scale are being met by growing limited quantities of "mother seed" in Wisconsin using heads carefully selected from "sick" soil, then sending this "mother seed" to the Puget Sound region for one generation increase. The output of seed is thus multiplied a thousand fold, insuring maximum commercial efficiency with minimum danger of loss of resistance. The Kraut Association is thus supplying the commercial needs in large kraut districts afflicted with yellows. The only danger foreseen is exploitation by some less scrupulous or intelligent commercial seed firms without strict attention to source of their mother seed.

*Blossom blight of the peach.* MEL. T. COOK

This disease, due to *Sclerotinia cinerea* (Bon.) Schrot. was much more severe in 1919 than usual. The crop on some trees was practically destroyed. The disease was much less severe on young than on old orchards, but there was no apparent connection with the mummified fruit or with varieties. The organism appeared to spread from the cankers on the 1918 wood. These cankers gave off gum and produced spores throughout the greater part of the growing season of 1919, but the fungus gradually became weaker and weaker. The cankers are evidently a very important source for the spread of the organism and apparently much more important than the mummied fruits. It is very doubtful if many of the cankers give off spores for more than one season. An important factor in control consists in preventing the formation of cankers. This can be done by more frequent and more thorough spraying.

*A heart-rot of peach.* W. A. MCCUBBIN

A rot of the heart wood of living peach trees, occasionally met with prior to 1915, became very prevalent in the Niagara Peninsula in Ontario in 1916 and 1917. Numerous cultures indicate *Coriolus versicolor* (L.) Quel. as the causal organism. It is claimed that the fungus most readily attacks wood injured by winter conditions, and this view is upheld by evidence showing the restriction of the rot to those portions of the trunk and limbs in existence before the very hard winter of 1913–14 and severely injured at that time; and further by the occurrence in many cases, of two regions of rot separated by a cylinder of sound wood, the outer region associated with tissues injured in the afore-mentioned winter and the inner region associated with wood affected by the winter of 1911–12.

*Changes of a decade in Plant Pathology.* F. L. STEVENS

A brief summary of major events, introduced diseases, progress in disease resistance, taxonomic changes, significant facts, new plant diseases, and losses of personnel through death.

*The development of Urophlyctis alfalfae.* FRED REUEL JONES AND CHARLES DRECHSLER

The disease of alfalfa caused by *Urophlyctis alfalfae,* commonly known as crown wart, has been found to have its origin from the infection of very young buds, the foliar elements of which develop into abnormalities not involving the mature structures of root or stem. The thallus of the fungus consists of a succession of two types of structures, turbinate cells and resting spores. In the first turbinate cell that is the immediate development of the infecting fungus, are inserted a number of septa which delimit uninucleated peripheral segments from a central polynucleated central sporogenous mass. A hyphal structure of limited growth develops from each of these segments and carries the nucleus in its expanded termination, the latter constituting a young turbinate cell of the next succession. At its mature stage the turbinate cell bears a branched apical haustorium, the short axial element of which proliferates at its tip a globose terminal expansion into which the polynucleate sporogenous mass of protoplasm migrates to produce the resting spore. This is characterized by 10 to 15 branched haustoria in zonate arrangement between the equator and the distal pole. A solution of the thinner cell walls in proximity to the young advancing turbinate cells leads to the development of cavities in the hypertrophied tissue in which the resting spores are finally found enclosed.

*Two destructive rusts ready to invade the United States.* J. C. ARTHUR

A rust on peanuts has long been prevalent in South America. In Trinidad and the nearby islands of the Antilles it is a great menace to the crop, often covering every leaf on the plants with a heavy powder of urediniospores. No successful method of controlling the rust has yet been found. A rust on potatoes and tomatoes, comparable in its life history and behavior with the hollyhock rust, has recently come to light in Costa Rica and Ecuador. The possibilities for its spread and harmfulness deserve serious attention. Neither this rust nor the preceding one has yet been reported from any United States possession.

### *Overwintering and control of bur clover leaf-spot.* E. F. Hopkins

Studies on the bur clover leaf-spot have shown that the causal fungus *Cercospora medicaginis* E. & E. is borne as mycelium on the seed within the burs. Seeds separated from the burs were found to contain a considerable number which showed dark markings on their surfaces. By disinfecting some of these seeds in mercuric chloride 1:1000 for thirty seconds, rinsing in sterile water, and planting in agar pure cultures of a fungus were obtained which in all cases appeared to be identical. Inoculations of healthy bur clover plants produced typical symptoms.

In attempting to determine the effect of boiling on the fungus with a view towards control, diseased seeds after being disinfected as above were planted in agar. It was found that boiling for as short a time as one-half minute would kill the fungus, while in the control cultures seeds which were disinfected but not boiled produced in each case a pure culture of Cercospora.

It is fairly certain from the above experiments and from microscopical examination that the mycelium occurs in the seed coats and that the fungus is not carried over, as Wolf has suggested (Phytopath. 6: 301. 1916), by means of viable conidia on the burs.

### *A disease of red clover and strawberry in the Pacific Northwest caused by the nematode Tylenchus dipsaci (Kuhn), Bastian.* L. P. Byars

The writer has observed in the Pacific Northwest during the past season a serious disease of red clover and strawberry plants caused by the nematode *Tylenchus dipsaci* (Kuhn) Bastian, which has long been known to produce considerable trouble of many different species of plants in Europe. Although known to have been in the Pacific Northwest since its discovery at Bellingham, Washington, in 1913, the nematode has not been previously known to cause serious damage in this country.

On strawberry the disease may be recognized by pronounced local swellings of the leaves, petioles, stems, and stolons or fruiting branches, which contain the causal organism in all stages of development. Affected plants are usually dwarfed and contain a large number of adventitious branches.

Severely infected red clover plants may be recognized by blister-like swellings of the leaves, petioles, and stems, and by the terminal bending of the latter. Such plants usually have a sort of witches'-broom appearance, due to the production of adventitious primary branches.

While the nematodes causing the troubles on these two hosts seem to be morphologically alike, there is no certainty that they are physiologically identical.

Investigations of the disease in laboratory, greenhouse, and field are actively under way.

### *Flax canker, a non-parasitic disease.* C. S. Reddy and W. E. Brentzel

This paper deals with the type of flax canker which occurs somewhat uniformly each year in the semiarid Northwest and causes a marked loss in flax production.

The cortex of the stem is killed at the surface of the ground. Sooner or later the plant topples over, but usually remains alive for days or weeks while the vascular system still functions. Usually the stem enlarges just above the injury and sometimes also just below. The result is a girdling of the plant at the soil line.

No parasitic organism has been found associated with this type of flax canker. Evidence seems to indicate that high temperature at the soil line during the early growth of the plant is the chief cause of the trouble. In preliminary experiments flax cankered only on and immediately following very hot days. It is susceptible only to a slight extent when more than four inches high. Plants shaded by a ten-inch vertical strip of canvas did not canker while unshaded plants in the same row cankered. Thickly sown flax cankered less than thinly sown flax. Flax sown with cereals cankered comparatively little, and flax with weeds cankered less than flax free from weeds. Killing the cortex of young flax plants by artificial heat produced typical flax canker.

*Pythium as a causal factor in "pea blight."* Fred Reuel Jones

The term "pea blight" as used by canners refers to several diseases of peas, but primarily to the retarded growth and death of plants consequent upon the decay of the stem below ground and more or less of the roots. In a number of localities in Wisconsin, Illinois, and Michigan selected for observation, where the disease is at its worst, several parasites have been found apparently contributing to produce this condition. Of these *Rhizoctonia solani*, a species of *Fusarium*, and *Pythium* (probably *debaryanum*) appear to be the more important. Under conditions prevailing in this region in 1919 Pythium appears to have been by far the most important, so that judged by this situation alone "pea blight" could be said to be primarily a Pythium problem. Under field conditions this fungus produces a typical "damping off" or wilt of occasional plants only, and in any case oospores have been rarely found in the diseased tissue.

*Phoma root-rot of celery.* C. W. Bennett

In several instances, in the past few years, a Phoma root-rot, caused by *Phoma apiicola* Speg., has been found doing considerable damage to celery in Michigan celery-growing districts. This disease attacks the roots and bases of the plants, resulting in a rotting off of the roots near the crown or in the production of large black areas just beneath the surface of the soil. Moisture is the controlling factor in the restriction of the pathogene to the underground parts, and lack of oxygen is important in keeping the roots, which are deep in the soil, free from this disease.

In culture, the causal fungus develops a bluish to mouse-colored mycelium when first isolated from the host plant. After growing on certain media for a period, color is no longer produced. Under similar conditions the organism loses both its power to produce fruiting bodies and its parasitic potentialities.

*Scientific instruments and patent rights.* Donald Reddick

It is proposed that new species of scientific apparatus for use in pathological work should be submitted to a committee of specialists for examination and test. When favorable reports are returned to the American Phytopathological Society Incorporated, the corporation should take out patent rights and market the product either by sale outright or on a royalty basis. This would insure that useful apparatus would be immediately available to all workers. The burden of the royalty no doubt would fall on the institutions, but it would be relatively insignificant. Any revenue from royalties would belong to the "Corporation" and might be used to further scientific research.

## *Studies on varietal resistance and susceptibility to bacterial blight of the soy bean.*

C. M. Woodworth and Florence Coerper Brown

Field observations and experiments at the University of Wisconsin during the past three or four years have indicated that soy bean varieties vary greatly in their relative susceptibility to the bacterial blight. Of 47 varieties secured from the Agricultural Experiment Stations of Illinois, Ohio, Indiana, Kentucky and Wisconsin, and grown in 1918, about half were completely resistant and the other half ranged from complete susceptibility to partial resistance. Experimental inoculations in the greenhouse have strengthened this evidence regarding varietal differences in resistance. Varieties Ebony, Elton, Habara, No. 8 (S. P. I. No. 20406), Mammoth yellow, Virginia, Cloud, Wilson, Medium yellow, and Ito San were under trial. All plants were sprayed with a water suspension of the bacterial blight organism shortly after the first compound leaf appeared. An examination made three weeks later showed about half of the plants of the Wilson variety, all but three of Medium yellow and all of Ito San, infected. The other varieties were completely resistant.

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# PHYTOPATHOLOGY

VOLUME X NUMBER 2

FEBRUARY, 1920

## A BACTERIAL LEAFSPOT OF VELVET BEAN

FREDERICK A. WOLF

WITH TWO FIGURES IN THE TEXT

During the summer of 1916, a leafspot of Florida velvet bean, *Stizolobium deeringianum* Bort, was noted in the vicinity of West Raleigh, N. C. It was apparently unlike any known disease affecting this crop, as judged from available published accounts. Furthermore, preliminary examination of these specimens showed that the affected areas were teeming with bacteria. Since the foliage of soybeans, *Soja max*, growing in the fields together with velvet beans was affected with a bacterial blight, it was thought at first that the causal organism on both host species was probably identical. Furthermore the colonies in the isolations on nutrient agar looked so similar that it did not become apparent that one was dealing with two distinct organisms until morphological studies were made and reciprocal inoculations were attempted. Parallel studies of the morphology, of the cultural and biochemical characters, and of the pathogenicity of the velvet bean leafspot organism and soybean bacterial blight, *Bacterium sojae*[1] have therefore been made, and the results of the studies on the former are given herewith whereas those on the latter are presented in another paper appearing in the March number of this Journal.

### THE DISEASE

The disease has been observed to be more or less destructive during the past four years, not only in the vicinity in which it was first seen, but specimens have also been collected in several widely separated portions of the state. In no case has it been sufficiently severe to be a limiting factor in the culture of velvet beans. Furthermore, since the variety

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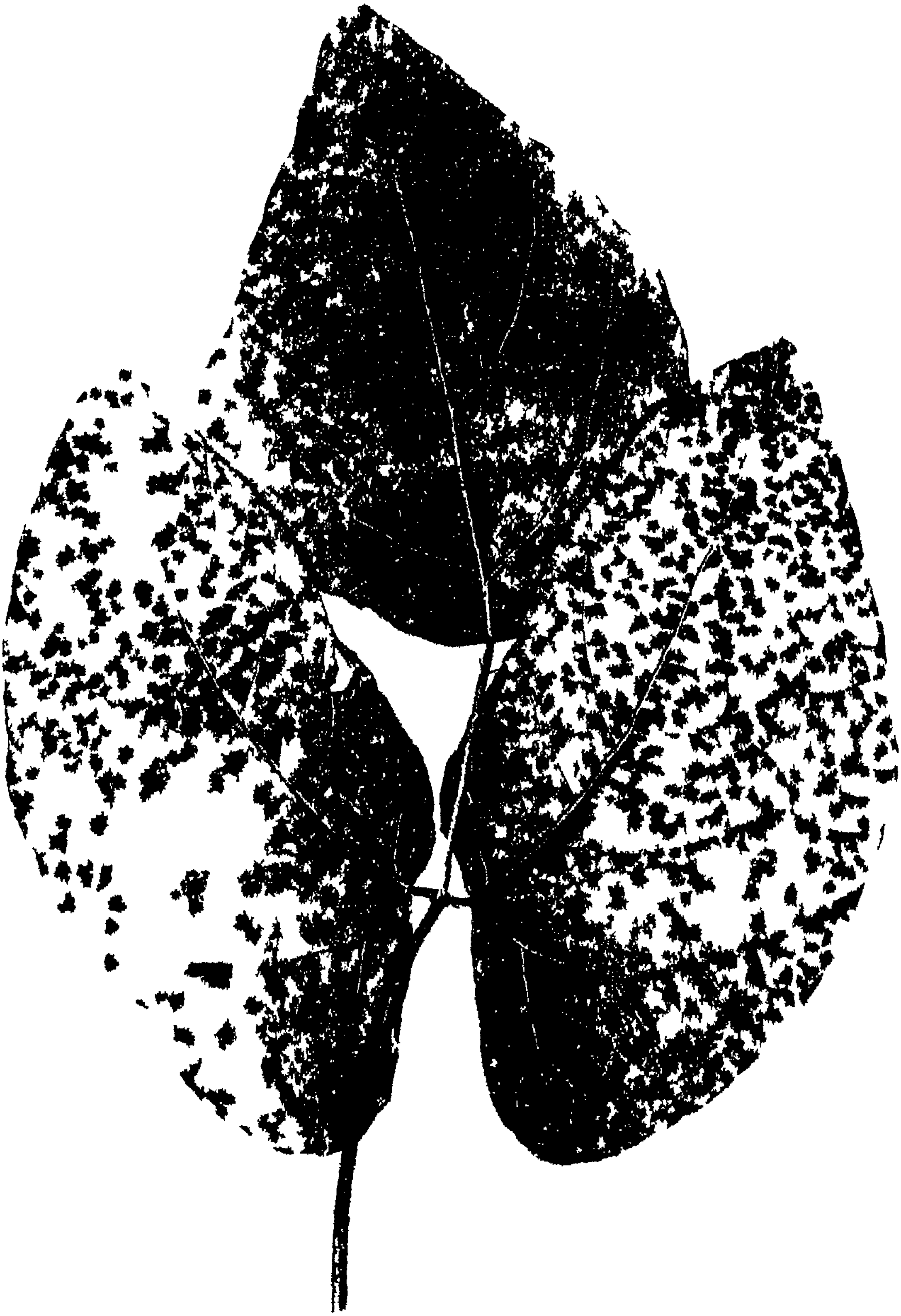

FIG. 1. LOWER LEAF SURFACE OF FLORIDA VELVET BEAN LEAF SHOWING THE APPEARANCE OF BACTERIAL LEAFSPOT

known as "Hundred Day Speckle" alone will properly mature in the latitude of North Carolina, no opportunity has been given to make field observations upon the susceptibility of Lyon bean, *Stizolobium niveum* (Roxb.) Kuntze, and Yokohama bean, *Stizolobium hassjoo* Piper and Tracy, species grown farther southward.

The disease may occur in fields which have never previously been planted to velvet beans, indicating transmission of the disease through seed. Apparently, however, the foliage only is subject to attack. Here the disease is first evident by the presence of small, point-like, translucent areas. The centers of these areas early become dark brown, and the translucent character largely disappears except at the extreme periphery. The water-soaked border, so characteristic of bacterial leaf spots, is entirely absent in mature lesions. An area near the center of mature spots is characteristically lighter in color than the margin. The tissues immediately surrounding the invaded areas are more or less chlorotic, and this chlorosis may involve all of the leaf surface not occupied by lesions. Mature spots are angular, being limited by the veins except when they are so abundant as to merge into large, irregular areas. They are commonly 2 to 3 mm. across, although they vary in size from mere points to spots 8 mm. in width. Severely diseased leaves may have as many as several hundred spots on each leaflet, as shown in figure 1. Little difference in appearance is to be noted between the symptoms presented by the upper and lower leaf surface, and no bacterial exudate is manifest.

## THE ORGANISM

The organism being non-motile is referred to the genus Aplanobacter, and since it is apparently unlike any previously described organism the specific name stizolobii is suggested. It is therefore described as *Aplanobacter stizolobii* n. sp.

## MORPHOLOGY

*Vegetative cells.* The organism is a short rod with rounded ends. It occurs singly or in pairs when taken from the host and may form short chains in nutrient broth. When taken from nutrient agar cultures and stained with carbol fuchsin it is slightly variable in length, being 1 to 1.6 by 0.6 to 0.7 $\mu$ (fig. 2, *b*). Mature elements average 1.3 $\mu$ in length and individuals exceeding this measurement appear to be undergoing fission.

*Endospores.* No endospores have been observed and little probability of their occurrence exists, in the light of the fact that the thermal death point is not far from 50 degrees C.

*Flagella.* Attempts to demonstrate flagella have been without success. Flagella of *Bacterium sojæ* are sharply differentiated by Morrey's modification of Loeffler's method, whereas no flagella are demonstrable on the velvet bean organism upon the opposite end of the same slide, where it is subjected to the same conditions of staining. When it is taken from young lesions and from 24-hour cultures on nutrient agar or in broth and examined for motility no evidence of independent movement can be discerned.

*Capsules.* The organisms used for this purpose were grown on potato plugs and were stained by the method of Rabiger and of Welch. A conspicuous envelope may be demonstrated by either of these methods (fig. 2, *a*).

*Zoogloeae.* Neither zoogloeae nor pseudozoogloeae are formed.

*Involution forms.* None observed.

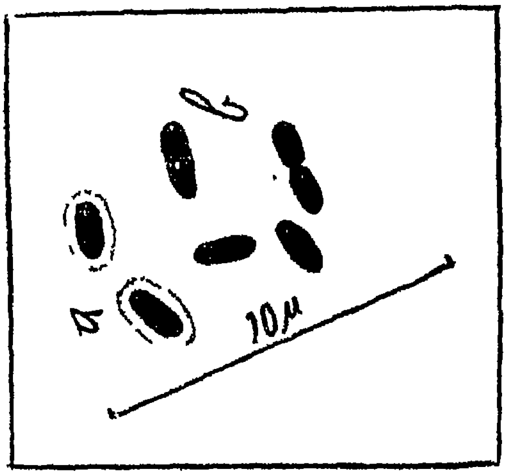

Fig. 2. *a*, Aplanobacter Stizolobii Stained by Welch's Method; *b*, Stained with Carbol Fuchsin

*Staining reactions.* Efforts at staining with the ordinary stains in both watery and alcoholic solutions have been uniformly successful. Gram's stain and Zeihl-Neelson's, however, proved negative.

## CULTURAL CHARACTERS

No particular difficulty has been experienced in isolating the parasite. Lesions from freshly collected material were cut out and thoroughly macerated in a drop of sterile water on a microscopic slide. A platinum loopful of this material was transferred to one edge of a hardened plate of nutrient agar, neutral to phenolphthalein. The material was then spread over the surface of the agar plate with a zigzag stroke in the ultimate direction of the side of the plate opposite to the one where the inoculum was deposited. The organisms are so distributed by this manner of spreading that well isolated colonies develop near the end of the stroke. The plate, of course, always contains species other than the parasite when this method of isolation is employed, since no attempt is made to disinfect the leaf surface before excision of lesions. The parasite uniformly predominates, however, and the danger of its destruction within the tissues, as so commonly occurs from the use of alcohol or bichlorid of mercury, is eliminated.

The peptone beef-extract agar and bouillon used in the cultural studies were enriched by the addition of either dextrose, lactose, glycerin, or saccharose to make 1 per cent solutions. The carbohydrate solutions were prepared separately and added to the media when the temperature had been lowered to about 60°C., and the media were not heated subsequently. All of the various media used in one series of cultural studies were inoculated on the same day from the same stock culture.

*Agar colonies.* When isolations are made from diseased tissue the colonies appear on the second day and are convex, semitransparent, circular, shining, and minute. By the fourth or fifth day at 20 to 25°C. they have attained their maximun size and are 1 to 1.5 mm. in diameter. They have a shiny luster, are smooth, white, raised, and opaque, and possess an entire or slightly undulate margin.

On agar slants there is a moderate filiform growth which spreads at the base of the slant where moisture of condensation is present. The streak is whitish and does not penetrate the agar nor give rise to any perceptible odor.

In agar stabs the growth is confined to the surface. It covers the greater portion of the tube in the course of a month, the edges are contoured, and there is no liquefaction of the agar.

*Gelatin colonies.* Stab cultures only, maintained at about 18°C., were used. There was no evidence of growth along the line of the stab and no liquefaction at the end of thirty days. The organism was able to make sufficient growth in this time, however, to cover the surface of the tube.

*Potato cylinders.* When the surface of the cylinders is very moist, growth is moderate, white, glistening, and spreading, whereas rather dry plugs have a raised, lusterless growth. The upper end of the streak will have become of a cheesy consistency within a month. No odor is developed within this period, the substratum does not become discolored, and there is no evidence of diastatic action when tested by means of iodin in potassium iodid solution. The organism is without effect on corn starch agar.

*Bouillon.* Peptone beef-extract bouillon becomes only slightly clouded throughout; no pellicle, ring, nor odor is developed.

*Litmus milk.* Freshly separated milk, without neutralizing, was made up to a violaceous color and subjected to four sterilizations in an Arnold sterilizer, at intervals of 12 hours. After ten days the blue color is increased to slightly darker than caeruleus. A considerable white precipitate appears within a month and the color is unchanged.

## PHYSICAL AND BIOCHEMICAL CHARACTERS

In the studies on reaction toward the several sugars, use was made of stab cultures on litmus agar and of peptone broth in fermentation tubes to both of which the appropriate carbon compounds had been added in the manner previously described.

*Litmus agar.* Neither acid nor gas were formed from glycerin, lactose, saccharose, or dextrose, although the cultures were incubated at about 20°C. for thirty days.

*Fermentation tubes.* Triplicate sets of fermentation tubes were prepared, inoculated, and kept under observation for three weeks. The open arm in all fermentation tubes became slightly clouded but in no case did growth extend into the closed arm, indicating strict aerobism. Neither was gas formed in the closed arm of any of the tubes.

*Nitrates.* Five-day-old cultures in 1 per cent potassium nitrate bouillon solution were tested for nitrites by the starch-iodide method. No blue color resulted, which indicates that there was no reduction of nitrates to nitrites. A solution of sodium nitrite used to test the reagents gave the characteristic reaction, however.

*Indol.* A few drops of concentrated sulphuric acid and 0.01 per cent solution of potassium nitrite were added to ten-day-old cultures in Dunham's solution. Even after the tubes were gently heated no pink color developed, indicating the absence of indol.

*Temperature relations.* The optimum temperature for the cultivation of this organism has not been carefully determined but appears to lie between 25 and 28°C. The thermal death point determined by exposing newly inoculated bouillon cultures for ten minutes to definite temperatures appears to be not far from 50°C.

*Group number.* Following the numerical system of the Society of American Bacteriologists the group number of this organism is 212.3332033.

## COMPARISON OF BACTERIUM SOJÆ AND APLANOBACTER STIZOLOBII

Since, as has been indicated, these two organisms looked so similar when the first isolations were made, parallel studies on one strain each, according to the essential features on the descriptive chart, were made.

The results of this comparison indicate that these strains differ strikingly only as follows:

| ORGANISM | SIZE | FLAGELLA | DIASTATIC ACTION | GROWTH IN BOUILLON |
|---|---|---|---|---|
| *Bacterium sojæ* . . | 1.2–2.6 by 0 8–0.9 $\mu$ | 1 to 4 | Very feeble | Surface pellicle and moderate clouding. |
| *Aplanobacter stizolobii*......... | 1–1 6 by 0 6–0.7 $\mu$ | None | None | No pellicle with less dense clouding |

### SUMMARY OF DESCRIPTIVE CHARACTERS

The results of the studies on the identity of the velvet bean leafspot organism may be briefly summarized as follows:

**Aplanobacter stizolobii** n. sp.

Elements cylindrical with rounded ends, single or in pairs, 1–1.6 by 0.6–0.7 $\mu$, non-motile, capsulate, no endospores, zoogloeae not formed. Neither acid fast nor stained by Gram's method.

Colonies on nutrient agar circular, raised, white, shining, margin entire or slightly undulate.

Gelatin not liquefied; casein partially precipitated and blue color in litmus milk intensified; nitrates not reduced; indol not formed; neither acid nor gas formed in cultures with various sugars; no growth in closed arm of fermentation tubes; no diastatic action.

Pathogenic on foliage of *Stizolobium deeringianum*, forming leafspots which are at first translucent and become at maturity dark brown.

Type specimens and artificially inoculated leaves have been deposited in the herbarium of the Office of Mycological Collections, Bureau of Plant Industry, Washington, D. C.

The brief Latin diagnosis is as follows:

Baculis cylindraceis, apicibus rotundatis, solitariis aut interdum geminis, 1-1.6 by 0.6–0.7 $\mu$, non-motilibus, capsulatis, sporis nondum visis; zoogloeis defectis; acidibus decoloratis, cum methodo Grami non coloratis; coloniis in agar-agar orbicularibus, convexis, albidis, nitentibus, marginis solidis aut lobulatis; gelatinis non liquefacientibus; casein ex parte segregantibus; lactem litmus non decolorantibus; nitrum non reducentibus; indol non producentibus; in mediis saccharatis neque gas neque acidum evolvatis; aerobicis; amylum non fermentatis.

Hab. In foliis vivis Stizolobii deeringiani, laesiones primum translucentibus diende-brunneolis efficiens.

### PATHOGENICITY

Florida velvet beans and Mammoth Yellow soybeans are the only host species which have been used in the inoculation experiments. It has been impossible thus far to bring about infection on the latter host with pure cultures of *Aplanobacter stizolobii*, but characteristic lesions follow inoculation of velvet beans. The following procedure and results are typical of the tests which have been made. A few leaves on one side of a large vine growing in the greenhouse were atomized with a forty-eight-hour bouillon culture late in the afternoon of September 11. On the fifth day following, numerous water-soaked lesions had formed which on the fourteenth day were characteristic of the disease. The abundance of the diseased areas on these artificially inoculated leaves can be judged from the following counts made on ten leaflets taken at random; 31, 215, 108, 35, 101, 175, 52, 96, 109, 30.

An organism which presents the same morphological features and appearance on agar as the original organism has been reisolated from artificial inoculations.

## PATHOLOGICAL HISTOLOGY

Four-day-old and two-week-old lesions from artificial inoculation, and mature lesions from field collections have been fixed, embedded in paraffin, sectioned, and then stained by Loeffler's methylene blue method. The presence of bacteria within the tissues may be clearly demonstrated in velvet bean lesions by means of this method. As in the case of a considerable number of leaf-invading bacteria, entrance is gained through the stomates, and in young lesions the substomatal cavities and adjacent intercellular spaces alone are occupied. As the lesions mature, however, and the invaded tissues become desiccated, the cell walls are disrupted, and the organism comes not only to occupy the intercellular spaces but to fill the interior of the cells. Apparently it is impossible for the organism to attack tissues other than parenchyma.

## SUMMARY

Bacterial leafspot of Florida velvet beans appears to be a hitherto undescribed disease, which has been under observation in North Carolina since 1916.

The disease is apparently restricted to the leaves, on which it is first apparent as translucent areas which become at length dark brown in color.

The parasite is unlike the soybean bacterial blight organism, with which it has been compared in essential characters by means of parallel cultural studies. It is herein described as *Aplanobacter stizolobii*, n. sp., is white in culture, and according to the numerical system has the number 212.3332033.

An incubation period of about four days follows inoculation of uninjured leaves. Infection occurs through the stomata; invasion is first intercellular but later becomes intracellular and only parenchyma tissues are involved.

# HEART ROT, RIB ROT, AND LEAF SPOT OF CHINESE CABBAGES

NELLIE A. BROWN AND R. B. HARVEY

WITH FOUR FIGURES IN THE TEXT

## GENERAL FEATURES

A rot which destroys a high percentage of the plants has been found prevalent on Chinese cabbage varieties in Maryland during the damp weather of last summer and continuing during the unusually warm autumn of this year (1919). The high temperature and wet season are no doubt the cause of the prevalence of the diseased condition. However, some growers have complained of this rot in more normal years and found the production of Chinese cabbages difficult on account of their susceptibility to the rot.

First indications of the disease are given by a sudden wilting of the plants although they are supplied with abundant moisture. In handling such plants it will be found that the head can be easily picked out from the crown of leaves, displaying a very rotten and foul smelling hole in the pith (fig. 1). The tissue within the ring of vascular bundles is usually soft and may be almost entirely removed. The vascular ring is browned. Flies and other insects are attracted by the foul odor, and probably play a part in dissemination of the disease.

A characteristic appearance which may occur accompanied or not by wilting is a browning of the midribs; this occurs noticeably in the looser leaved varieties, Pak choi, *Brassica chinensis* Jusl. Besides this very marked browning of the midrib, specking or spotting of the leaf blade may occur. These spots can be distinguished readily from spots on the leaf caused by *Alternaria brassicae* (Berk.) Sacc., which are round or oval, black spots marked with concentric zones. The Alternaria spot (fig. 2) generally does not cause as much injury as the heart and rib rots, since it attacks only the outer leaves and is more amenable to control by spraying.

All varieties of Chinese cabbage which were tested, including Paoting, Peking, Kinshiu, Chosen, Shantung, and Santo Sai, are subject to this bacterial rot, but it seems more prevalent on the firm-headed varieties, Paoting and Peking of the Pai Ts'ai type. Loose-headed varieties of the

Pak choi type are less susceptible, probably because less moisture collects in the head or owing to the toughness of the epidermis.

When the crown of a diseased plant is cut across it is found to be hollow, the pith gone or nearly so, with the brown-colored vascular tissue left in a sort of honeycomb arrangement. The main and lateral roots are affected similarly, many of them being a brown color throughout. There is usually no soft rot condition of the main root or lateral roots, their affection being a dry rot. The soft rot occurs in the crown and bud, which become a slimy mass. These soft parts are well invaded by nematodes, fungi and bacteria.

The browned midrib is a dry rot (fig. 3) though it is not unusual for the soft stage to occur here also.

### THE CAUSE OF THE DISEASE

After repeated poured-plate isolations from different parts of the plant and from several plants, and inoculations into Chinese cabbage plants, we conclude that the beginning stage of the disease is produced by *Bacterium campestre* (Pam.) Smith, an organism producing the black rot of common cabbage and other cruciferous plants (3), (5), (6). This disease affecting the Chinese cabbages, which are succulent plants and rapid growers, produces numerous lesions which give opportunity for the entrance of a soft rot organism. The latter organism we found present in all the soft parts of the diseased plants. It is not present in the brown midribs unless the soft condition obtains, nor in the early stages of the dry rot condition of the lateral roots.

This soft rot organism seems to be the more destructive of the two (1). While *Bact. campestre* is slowly plugging up the vessels and dissolving the middle lamellae, causing wilting, formation of cavities, and brown staining, should the soft rot germ gain an entrance and be given the proper temperature and moisture conditions its destructive action is so rapid that it can do its work in advance of the wilting and browning. It produces a heart rot which spoils the plant entirely for either sale or home use.

### THE PARASITE

The initial cause of the disease is a short, yellow rod with one polar flagellum, motile but not always so. The rods are usually one and one-half to three times longer than wide, while sometimes they are so short that they resemble coccus forms. The organism most frequently occurs singly, but it occurs also in pairs and occasionally in chains of 4 to 12 or more of the very small rods. No spores were seen.

These morphological features are exactly like those of *Bact.* isolated from the common cabbage.

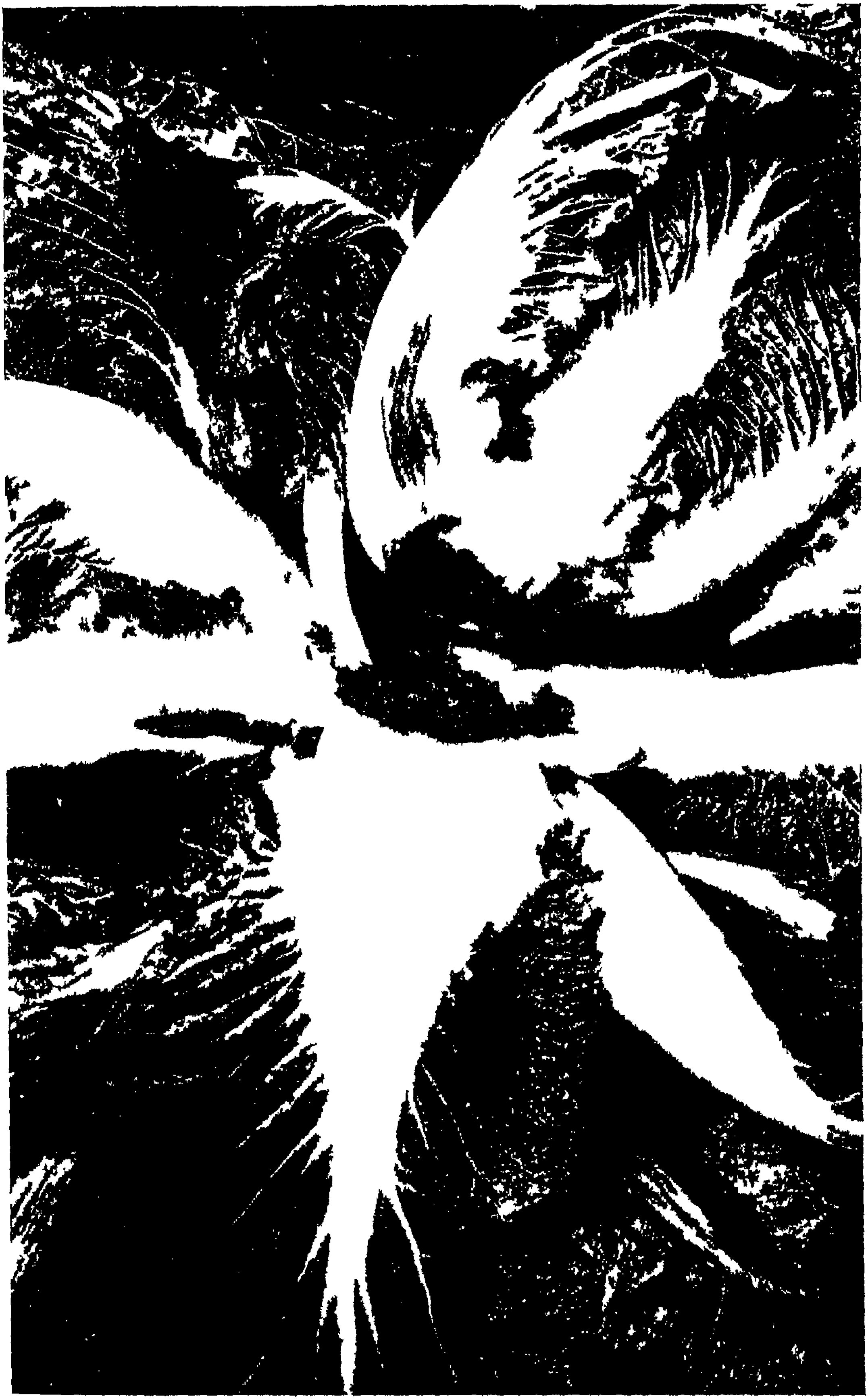

Fig 1 Chinese cabbage disease showing browning of midrib produced by *Bacterium campestre* and soft rot at heart produced by a soft rot organism

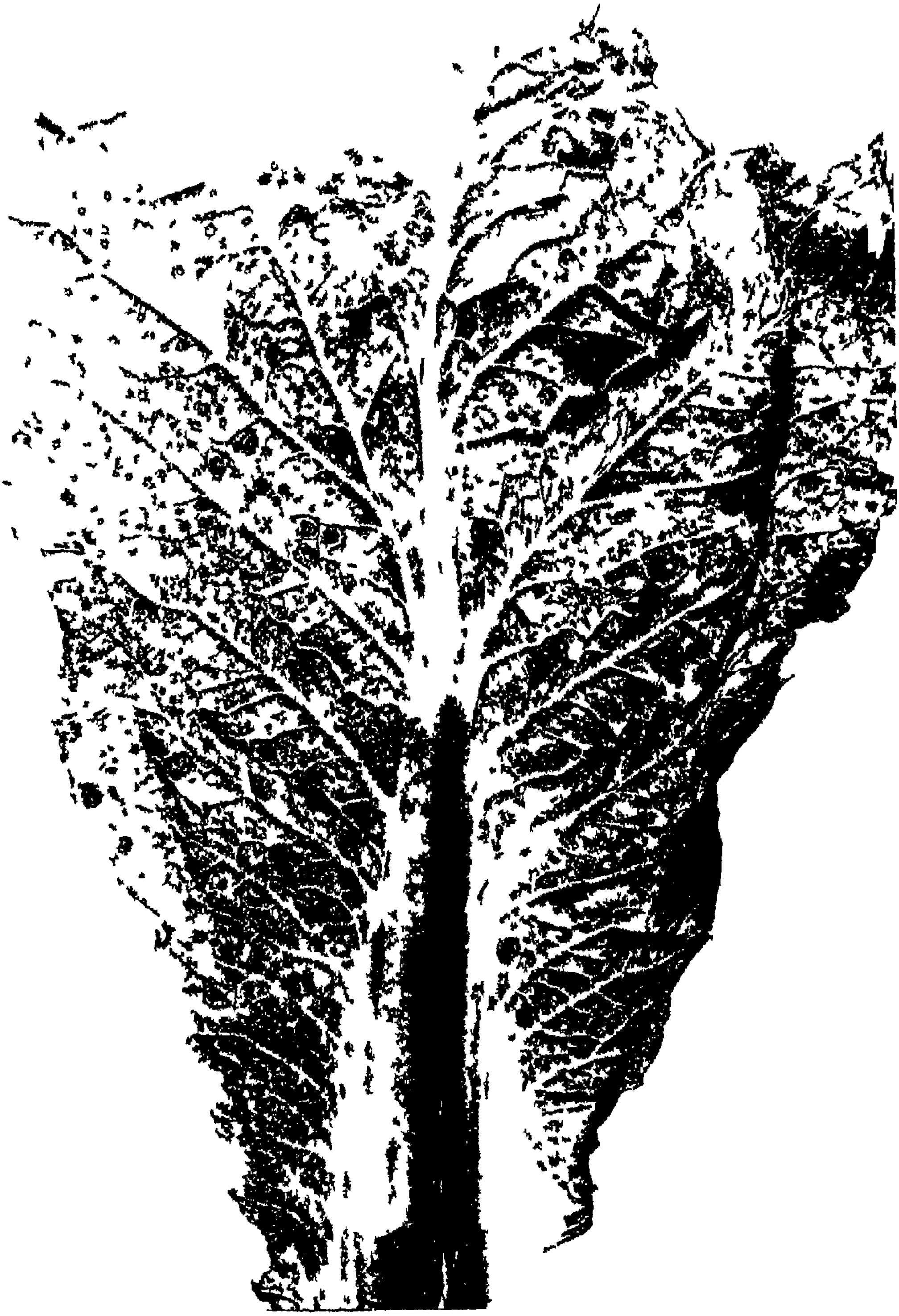

Fig 2 Chinese cabbage leaf showing browning of midrib and small dark spots produced by *Bacterium campestre* also zoned fungous spots of *Alternaria brassicae*

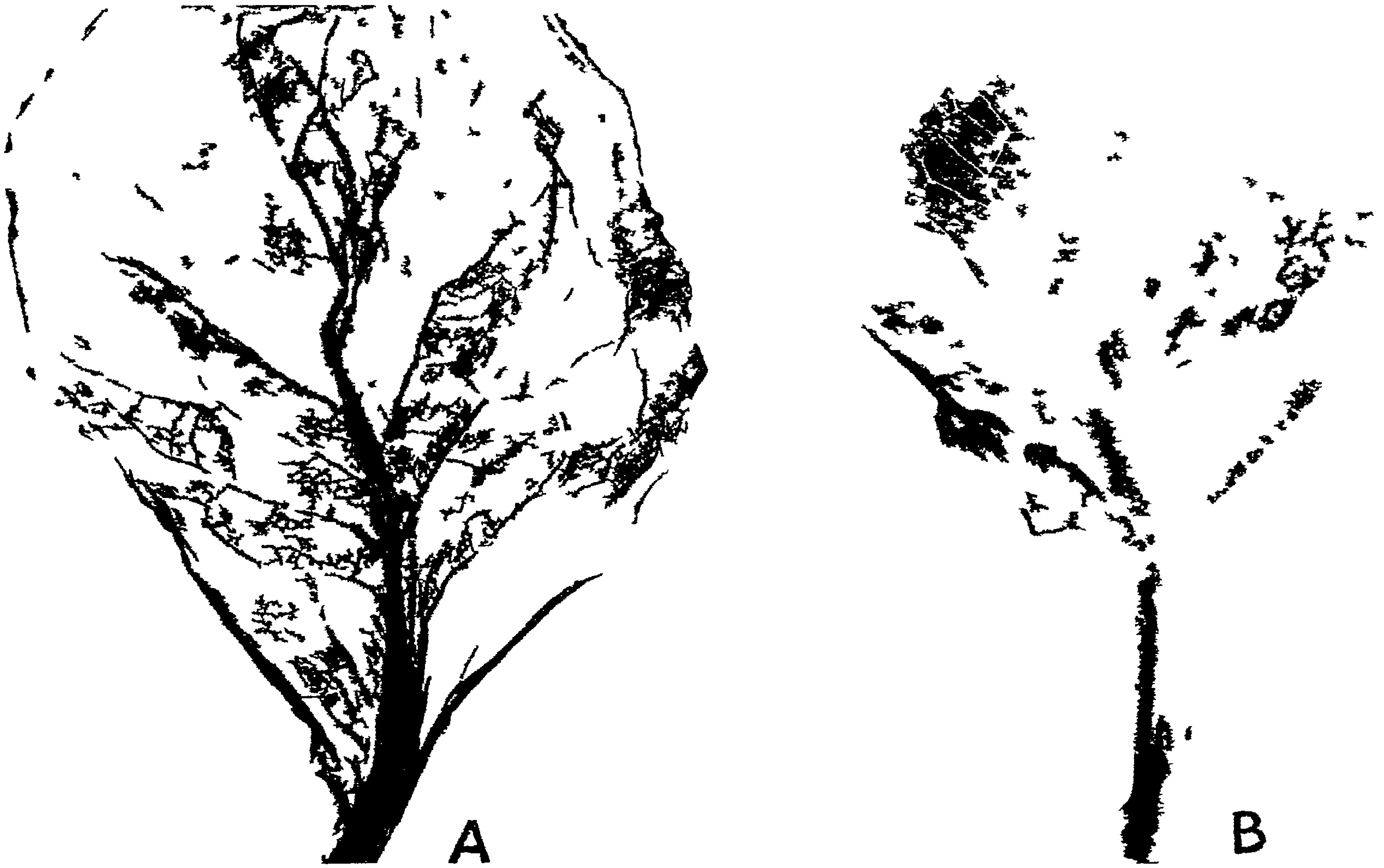

Fig 3 *A* Common cabbage leaf inoculated at base of midrib with *Bacterium campestre* isolated from Chinese cabbage Photographed seventeen days after inoculating

*B* Common cabbage leaf inoculated at base of midrib with same cultures Photographed two weeks after inoculating

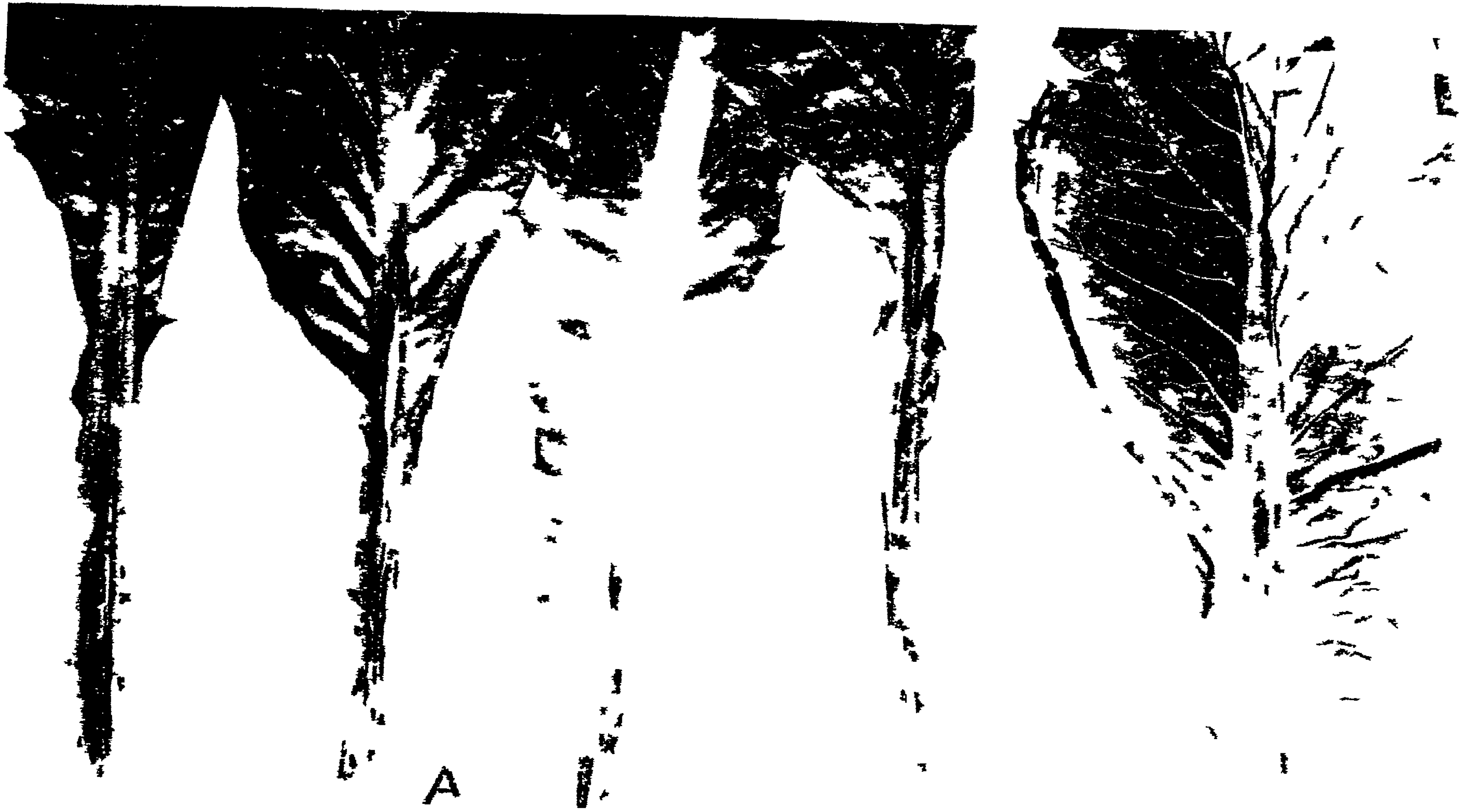

FIG. 4 4. Chinese cabbage leaves inoculated at base of midrib with *Bc* [illegible] isolated from Chinese cabbage. Photographed seventeen days after inoculating.

*B*. Chinese cabbage leaves inoculated at base of midrib with same isolation. Photographed nearly four weeks after inoculating.

## INOCULATIONS

Inoculations were made into Chinese cabbage and common cabbage by spraying the plants with water solutions of the organism made from agar cultures; also by smearing the bacteria on the leaf surfaces and stems and puncturing with a sterile needle.

Infection was slow in the sprayed plants, two to three weeks being required. When the surfaces were smeared and punctured, infection took place in from four days to a week and continued to spread.

Infection has two lines of travel from the water pores or other points of infection on the leaf down the blade to the stem; and when the stem is infected first through the fibrovascular bundles of the stem out into the midrib and blade. In the field, most of the infection seemed to be from the stem and roots or base of the leaf upward.

Fully 90 per cent of the inoculations took. The organism was reisolated from the browned midribs of the Chinese cabbage. There is some difference in the appearance of the infection of the two species of cabbage. In the Pai Ts'ai and Pak choi the brown veining does not show in the blade so definitely as it does in the common cabbage (figs. 3 and 4), nor is the discoloration so dark in the Chinese cabbages as in the common cabbage. However, the dark brown midrib in the Chinese cabbages, which may be an inch or more broad, is a very striking feature. Inoculations into the stem by punctures caused withering of the plant in two to four weeks, depending on the age and rate of growth of the plant.

The temperature of the greenhouse was around 78°F. day time and 65°F. at night, and the plants had plenty of moisture but not too much.

One set of puncture inoculations was made in a cooler house kept at 70°F. day time and 55°F. at night, and the amount of moisture reduced. At the end of a week there was no trace of infection in the plants at the lower temperature and reduced moisture, while those plants inoculated in the same way at the same time and kept in the warmer house with more moisture supplied showed good infection at the end of the week.

One set of inoculations was made by placing drops of a water suspension of the organism very carefully in the margins of the leaves of quickly growing plants of both Chinese and common cabbage. Darkening of the tiny veins was noted in the common cabbage in seven days but not until twelve days in the Chinese cabbage. The course of the disease continued to be more rapid in the common cabbage.

### CULTURAL CHARACTERS

The causal organism isolated was at first thought to be a new one, as its appearance on the beef-agar plates did not seem to agree with the description of *Bact. campestre*. There are other cultural differences between it and *Bact. campestre* isolated from the common cabbage (1), (7) but these differences are not sufficient and the pathogenic features are enough alike to decide that we are dealing with *Bact. campestre* infectious to new species of Brassica, namely *Brassica pekinensis* (Lour.) Gagn. and *Brassica chinensis* Jusl.

The colonies on potato agar appear two to three days after pouring plates; they are at first cream colored; when a day old they are yellowish in reflected light, a little lighter colored in transmitted light, with fine markings on the surface. They are round, thickish, smooth shining, 2 to 4 mm., four days after pouring from diseased material. The youngest ones have no ring margin; the older ones a definite ring margin which shows radiate lines under the hand lens. The margin which is thinner and lighter colored than the rest of the colony is in some cases almost bluish. As the colonies get older they become a definite yellow color and some of them are a centimeter in diameter.

On beef agar the colonies are at first cream white, bluish in transmitted light, many with a ring margin. They become yellow as they grow older.

Sterile milk is cleared without coagulation, the casein being precipitated slowly, the clear whey becoming a light yellow color.

Litmus milk is blued slowly; in sixteen days it is nigrosin violet (Ridgway).

The organism has a distinctive growth on sterile potato cylinders. It is bright yellow at first, filling the water in the tube with a dense, yellow slime. Later the growth becomes a brown color. There is slow breaking down of the starch.

Beef gelatin is liquefied in two weeks.

No definite pellicle is formed in beef bouillon. There is no growth in Cohn's solution, no reduction of nitrates to nitrites, no production of gas or of indol.

The organism, unlike *Bact. campestre* isolated from the common cabbage, will grow in beef bouillon over chloroform. It is unlike it also in that it produces no indol while the common cabbage organism produces a slight amount; also in its negative action in Cohn's solution, for the common cabbage organism sometimes grows in Cohn's solution.

However, these cultural differences are no more significant than those differences often found in separate isolations of the same organism from the same species. Here we have isolations from two different species.

### TREATMENT

The treatment of the disease falls into the class of those diseases in which preventative measures have to be taken. The treatment of seed is recommended. It will have little value if the soil is infected, but less risk is run if the start is made with clean seed.

*Bact. campestre* is not a spore bearer but the vegetative rods seem able to survive long periods of drying.

Harding, Stuart, and Prucha in their work with common cabbage proved the presence of *Bact. campestre* on four lots of cabbage seed. Four black-rot infected seed-cabbage plants were threshed separately, the seed rinsed in sterile water, and cultures made. *Bact. campestre* was obtained in the cultures from all four lots and its pathogenicity proved by inoculations into cabbage plants.

They conclude that much of the seed on the market is contaminated with *Bact. campestre* and that some of these germs may survive the winter and infect the young cabbage plants that are produced in the spring. They recommend the soaking of seed fifteen minutes in a 1–1000 corrosive sublimate solution or in formalin 1 pound to 30 gallons of water.

It has been shown by their experiments that cabbage seed soaked in water to which a culture of *Bact. campestre* has been added, then the seed dried and put away in test tubes, had live bacteria present eleven months afterward (8). These bacteria produced the characteristic symptoms when inoculated into cabbage plants.

In the seed bed, soil should be used that is known to be free from disease. Otherwise, it should be steamed in advance or a fire built over it.

As moist conditions play an important part in the appearance and spread of this disease, a fundamental requirement in growing the Chinese cabbages is a well drained field.

Worms and insects are disseminators and they can be reduced in numbers by spraying with insecticides.

Infection from the soil has to be considered, for this can occur readily with rough handling of the seedlings at the time of transplanting. Should the organism be present in the soil, then the injured roots with exposed fibro-vascular bundles offer a ready entrance for the bacteria and root infection follows.

The removal of infected leaves from the plant even when the disease first makes its appearance in the field has not proved a successful method of combating the disease (2).

Care taken in the destruction of diseased plants will go a long way toward the prevention of further losses another year. Infected plants should never be left to decay but should be burned or thrown into a jar containing dilute, crude sulphuric acid, or mixed with fresh lime.

Thoughtless dumping of diseased plants on the compost heap to be used later for fertilizing may account for infection in new fields. It is not only gardeners, ignorant of the infectious nature of plant diseases, who do not practice sanitary methods with plants, but often those who are informed. Field sanitary methods cannot be emphasized too much.

## SUMMARY

The Chinese cabbages—both loose and tight-headed varieties—are subject to the black rot disease of the common cabbage produced by *Bact. campestre*, and like the common cabbage there may be also a rapid secondary infection by a soft rot organism.

The disease is very destructive in those seasons when there are high temperatures with excessive moisture.

The organism enters the plant through the water pores of the leaf and through the wounds in roots and stem. Insects also act as carriers.

The broad midribs of the Chinese cabbages turn a dark brown. Innumerable spottings may occur in the blade.

The soft rot organism destroys the heart at once. The spotting of the blade is unlike that due to the fungus *Alternaria brassicae*, which is a spotting confined to the loose outer leaves of the head.

Treatment is of a preventive character. Sanitary methods in the fields where the disease has occurred, spraying to destroy worms and insects that might act as carriers, careful handling of plants while transplanting them, and treatment of seed with corrosive sublimate 1--1000 for fifteen minutes or formalin 1 pound to 30 gallons of water are recommended.

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# A NEMATODE DISEASE OF RED CLOVER AND STRAWBERRY IN THE PACIFIC NORTHWEST

L. P. BYARS

WITH PLATES VI AND VII

During the past summer the writer has observed a serious disease of red clover (*Trifolium pratense* Linn.) and strawberry (*Fragaria* sp.) plants in the Pacific Northwest, caused by the nematode *Tylenchus dipsaci* (Kühn) Bastian, (synonym *T. devastatrix*) which has long been known to attack many different species of plants in Europe but which has only recently been discovered to cause very severe damage in this country. Since the discovery of this nematode on hyacinths at the bulb propagating garden of the United States Department of Agriculture at Bellingham, Washington, in 1913, by the writer (1), Dr. N. A. Cobb[1] has observed it each successive year on specimens of red clover or strawberry plants sent in from other localities in the Northwest. During the past season Prof. Ralph. H. Smith (5) of the University of Idaho has found this parasite to be the cause of heavy losses to the red clover crops in many localities in Idaho, and it is also known to occur on this crop in eastern Washington and Oregon and in central Utah. In June of this year, the writer observed the nematode attacking strawberry plants growing on the experimental farm of the Oregon Agricultural Experiment Station, Corvallis, Oregon, where it had been seen three years previously by Prof. A. L. Lovett.[2] Since this article has been prepared the author has found the nematode on strawberry plants from Port Terrace, Oregon. These plants were collected in July, 1915, and sent to Dr. C. L. Shear, who preserved them in formalin and who kindly submitted them to the writer. Outside of the Pacific Northwest the nematode is known to have occurred in 1907 in Kansas, where Dr. E. A. Bessey (2) discovered it causing a stem disease of rye. Very probably it occurs in other parts of the United States and has been overlooked.

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[1] Cobb, N. A., U. S. Department of Agriculture. Unpublished data.

[2] Lovett, A. L., Oregon Agricultural College. Letter to Dr. N. A. Cobb, dated July, 1916.

## DESCRIPTION OF THE DISEASE

*On red clover.* As seen in irrigated fields of Idaho and Utah, mature diseased plants are usually reduced in size and send up from the crown a number of adventitious shortened stems or shoots, which gives to badly affected plants somewhat of a witch's-broom or bush-like appearance, as shown in plate VI. The crowns of such plants generally show a dry rot, which extends for a short distance downward into the tap root and upward into the stems. Because of this internal rotting, the above-ground parts of these plants may be easily separated from the tap root by slight pulling. The stems are often swollen, particularly near their bases, may have their internodal spaces shortened, and are frequently bent near the end. Affected leaf stipules, petioles, and sometimes the leaves become distorted and show blister-like swellings. Badly diseased plants usually lose their normal green color, wilt, and die. However, there seems to be no characteristic discoloration of the foliage associated with the earlier stages of the disease.

*On strawberry.* Manifestations of the disease on strawberry may be seen on any part of the plant above ground. The plants as a whole are usually somewhat dwarfed, of a lighter green color than normal plants, and produce at the crown many adventitious branches. As shown in plate VII, affected leaves are distorted, wrinkled, swollen, and may be greatly reduced in size. On stems, petioles, stolons, fruiting branches, and at the base of leaves conspicuous galls which contain the nematodes in great numbers may be produced. This disease is different from the so-called "cauliflower" disease of strawberry described by Ritzema Bos (4) and caused by another nematode, an *Aphelenchus* sp., presumably *A. ormerodis* Ritzema Bos. Plants attacked by the latter nematode, which is much smaller than *Tylenchus dipsaci,* produce a large number of dwarfed adventitious branches, some of which are stimulated by the nematode to grow together throughout their length. Because of this stem thickening and fasciation, diseased plants resemble somewhat the upper portion of a cauliflower or pineapple plant. So far as known an *Aphelenchus* sp. has not been reported on strawberry in this country although one has been described on violets and other hosts.

## DISTRIBUTION AND DAMAGE

The disease on red clover has been found only in the Pacific Northwest and in this region only in the irrigated sections. Whether it is confined to clover that is grown under irrigation is an important question which should be answered as soon as possible. The writer has been unable to find it on small plantings of clover in the neighborhood of Greeley, Colorado, Madison, Wisconsin, or Washington, D. C., but has not had opportunity to make even minor observations in other sections east of the Pacific Northwest.

Microscopic examinations by Dr. N. A. Cobb, Prof. Ralph H. Smith, and the writer show that the causal nematode in a living condition can be transported along with cleaned and uncleaned clover seed. It is highly probable that the parasite has been introduced into and spread within this country by this means. Spread in the irrigated regions no doubt has been largely by water.

General infestation of the clover in some of the irrigated sections of the Northwest has already taken place. This is particularly true in the Twin Falls, Idaho, district, where it is difficult to find an uninfested field and where the damage has been so severe that many farmers the past season have plowed up their clover and planted other crops.

Although preserved specimens of the disease on strawberry, collected at Port Terrace, Oregon, have been seen, the trouble on this plant has been found in the field at only one point, Corvallis, Oregon. At the latter place none of the plants were dying from the trouble at the time of examination although much damage in the way of reduced yield and lessened quality of fruit was apparent. On strawberry the trouble is very probably most commonly spread by means of the young infected plants which are used for propagating purposes.

## CAUSE

The disease of both the red clover and strawberry is caused by the nematode *Tylenchus dipsaci* (Kühn) Bastian, which usually attacks only the above-ground parts of plants. The eggs, larvae, and adults of this organism in great numbers may be seen with the aid of a hand lens if infected tissues are teased apart in a few drops of clear water. The adults are spindle-shaped, milky-white eelworms of little more than 1 mm. in length. In a desiccated condition the parasite, in its earlier stages of development, may remain motionless but alive for more than a year.

Often closely associated with the nematode on clover are certain other organisms, which seem to assist in only a secondary manner in causing the trouble. These are a small mite and the larvae of two insects, which were found constantly about the tap root and crown of affected plants and which have been previously thought to be the primary cause of the trouble. The writer has, however, just completed inoculation experiments in the greenhouse and laboratory which prove that the nematode alone is responsible for inducing the disease on clover. Details of the results of these experiments, which are being continued, will be published later.

Because of a lack of opportunity, inoculation experiments designed to demonstrate that the nematode is the sole cause of the disease on strawberry have not been carried out, but on affected plants in the field no other parasite could be found.

## HOST RELATIONS

No consistent morphological differences between the nematode on strawberry and red clover could be found by the writer. That there may be physiological differences, however, seems probable since rows of strawberry plants free from infection were observed growing in fields of clover which were severely diseased. Likewise disease-free crops of beans, alfalfa, and rye, which have been reported as hosts in Europe, were seen in fields that had grown a severely infected crop of clover the previous season. These observations harmonize with the view held by Ritzema Bos (3), namely, that in Europe this nematode becomes specialized on certain of its hosts and will not readily attack any other. The possible development of biological strains of the organism is an important and interesting question which must be solved before practicable methods of control can be well devised or carried out. Until this has been accomplished it would seem inadvisable to plant on infested land any of the crops which are known to be seriously damaged by the nematode in Europe. Some of the more important of these are: beans, potatoes, onions, peas, alfalfa, and most of the clovers, vetches, and grains. The writer has found the nematode attacking only one wild plant, a *Physalis* sp., which was growing in a badly diseased field of clover in Utah. Plants of this genus apparently have not been previously reported as a host (5).

Active investigations of the disease are under way in the laboratory and greenhouse, and field studies in coöperation with the Idaho Experiment Station have been planned.

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## PLATE VI

A NEMATODE-INFECTED RED CLOVER PLANT FROM PROVO, UTAH

Photographed natural size. Note the adventitious, dwarfed branches, some of which have swollen terminals.

Byars Nematode Disease of Clover and Strawberry

## PLATE VII

A NORMAL (ABOVE) AND A NEMATODE-INFECTED (BELOW) LEAF OF STRAWBERRY FROM CORVALLIS, OREGON

Photographed natural size. Note on the diseased leaf the distorted, dwarfed leaflet and the nematode gall on the petiole.

# STREAK DISEASE OF POTATO

W. A. ORTON

WITH PLATE VIII AND ONE FIGURE IN THE TEXT

In the summer of 1912 the writer studied a disease of potato that occurred in Maine, New York, and Wisconsin, and prepared a description which was intended to be published under the name "potato streak" in a bulletin on "Potato Wilt, Leaf-Roll, and Related Diseases."[1] This article was withheld because the nature of the streak disease had not been demonstrated and it was hoped that further laboratory work would result in the discovery of a parasitic causal organism. Our efforts in this direction were unsuccessful, and it now seems desirable to record our early observations, defining the characteristics of this interesting potato malady, which is being found from time to time in various parts of the country. There are somewhat similar obscure troubles which it is desirable to differentiate from the streak for clearness in discussion, even though it is possible that future research will reveal a relationship.

Potato streak is a disease of the foliage, attacking full grown and otherwise normal and healthy plants and frequently progressing rapidly to destroy them.

## CHARACTERISTICS

So far as our observations go, streak is first to be detected on the upper, full grown leaves of the potato plant, in the form of elongated or angular spots following the veinlets and invading the parenchyma (fig. 1). While somewhat more conspicuous on the upper side, these spots are also to be seen on the under side as narrow, discolored streaks along the veins. The typical form may very quickly be recognized and distinguished from the spots caused by early blight (*Macrosporium solani*), though the more diffuse spots of the streak resemble slightly the Macrosporium spots, particularly when the latter also occur on the same plants, as is often the case.

The destructive progress of the streak is rapid. Shortly after its appearance on the leaflets, as described, the petiole will be found affected and slightly discolored by longitudinal streaks. The petiole collapses and the leaf then withers and hangs limp, or the petiole breaks at the point of attachment to the stem and hangs by a thread in a dead and hued condition. Since it is the full grown leaves that are first attacked there is at this stage a circle of dead leaves about a third of the way down from the terminal shoot while the latter is still green (plate VIII, fig. 1).

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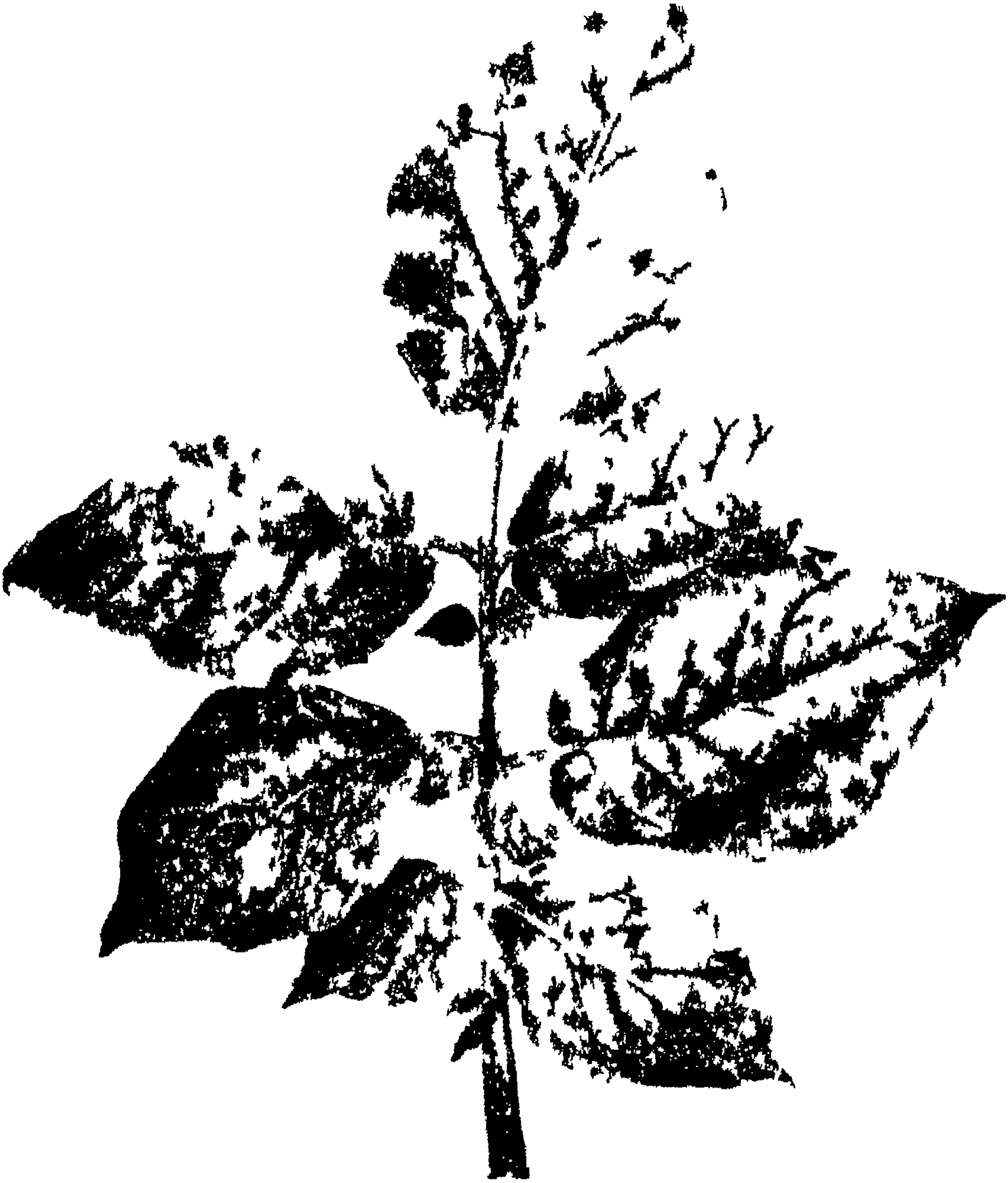

Fig. 1. Potato Streak

Drawing from nature of the early stage of streak from experimental plots in northern Maine. J. I. Brewer

The hanging dead leaves are quite characteristic of the disease while all the characters mentioned form a picture that is quickly seen and well remembered.

A prominent characteristic of streak is the brittleness of the affected parts. The leaves break off very easily and the stem is also brittle. This symptom is, however, also met with in other potato diseases, such as curly-dwarf and mosaic dwarf.

The stem begins to turn brown and dies at a point below the tip. The upper leaves then wilt and die, and the disease progresses downward. Long, faint brown streaks appear on the stems also. Apparently the discoloration is just below the epidermis but not in the vascular bundles. These brown stripes are not continuous from leaflet to petiole, nor from petiole to stem. There is no discoloration of the vascular bundles in the lower part of the stem or root, nor are there any other evidences of disease at the root. The several stalks in a hill die separately; all stages may be observed in one hill, from the first spotting of the leaves to quite dead stalks. A late stage is illustrated in plate VIII, figure 2.

No effect on the tubers has been found. The yield is reduced in proportion to the time of onset of the disease. The first cases were found when the Maine field was visited, August 9, 1912. At this time several plots were dead and the disease was quite generally distributed. The New York field was visited August 16 and 17, at which time the disease was under full headway. A considerable portion of the field was dead, and as all stages of streak could be traced, it was concluded that this new disease was the principal cause of the early death of the foliage, particularly in the seedling collection of the Bureau of Plant Industry which was being grown in both states. In other parts of the field there was a good deal of tipburn and early blight.

### VARIETAL RESISTANCE

Marked differences in varietal susceptibility exist. This was most prominent in the collection of seedling varieties where streak was most prevalent. Some plats remained entirely healthy, while others directly alongside were destroyed. More streak has been observed in European varieties, such as Factor, than in American sorts. The seedlings referred to were for the most part hybrids with European varieties of potatoes, and this may account for their greater susceptibility. Outside of this seedling collection, the most prominent case of streak the writer has observed was a field of several acres of Factor at Puyallup, Washington, in September, 1914. It was observed that year at Jerome, Idaho, but this also in seedling varieties. A very typical case was observed in a field of Rural New Yorker near Madison, Wisconsin, in 1912.

The nature of the streak disease remains in doubt. Its appearance and general course present striking similarities to some of our well known bacterial diseases, such as *Ps. malvacearum* on cotton and *Bact. michigensis* on tomato.

The writer was confident that bacteria would prove to be the cause, but efforts to isolate an organism have thus far been futile. For the most part plates poured from the discolored areas remained sterile. Colleagues have made similar attempts, with similar negative results. It is believed, nevertheless, that eventually an organism will be found and shown to cause potato streak.

## OTHER CONFUSING LEAF-SPOTS

It will be noted that the illustrations presented show streak on plants of normal size and development, and this is typical of the disease as the writer knows it.

It should not be confused with the spotting and streaking of potato leaves associated with advanced stages of leaf-roll or mosaic dwarfs, or from weak or otherwise degenerate plants. Such spots and streaks are common on moribund potato plants, but the pathological picture is quite different from that described above.

There is, for example, a disease not uncommon in our trial grounds, marked by weak, erect stems, from which frequently the spotted leaves fall prematurely, from the base upward, so that a palm-tree effect is produced.

It is quite within the possibilities that streak may attack a plant suffering from mosaic or other disease, and that when we know the cause of streak we shall be able to differentiate it more clearly from other leaf spots. The point to be emphasized now is that streak is not per se the result of some other disease, but is quite distinct and separable from all other potato diseases known to the writer.

To one observing the rapid spread of this disease through plats of susceptible varieties and its destructive effect, it would appear potentially dangerous to the potato industry, but seven years have passed since it was first observed, and the disease is still something of a rarity. This may be taken to indicate that our standard varieties of potatoes possess substantial resistance to streak and that it is not to be greatly feared by commercial growers.

BUREAU OF PLANT INDUSTRY
WASHINGTON, D. C.

## PLATE VIII

FIG. 1. Potato plant, unnamed seedling variety, dying from streak. Note dead leaves hanging in center where streak developed first.

FIG. 2. Potato streak, advanced stage. This plant was normal and healthy two weeks earlier.

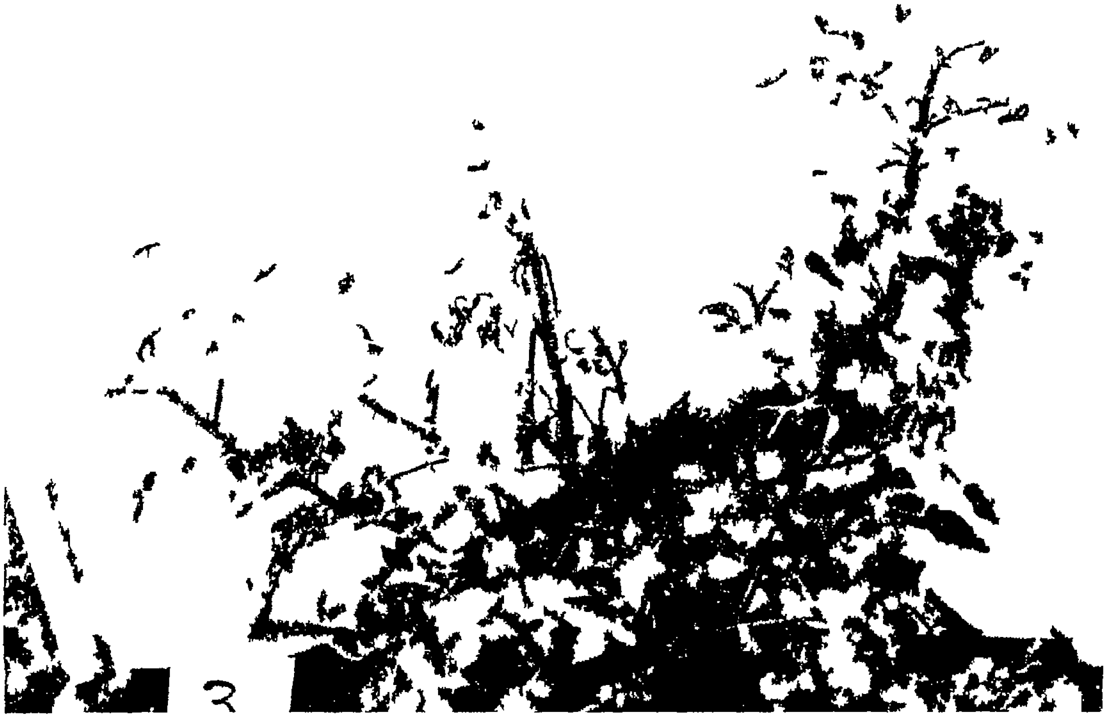

Fig. 1

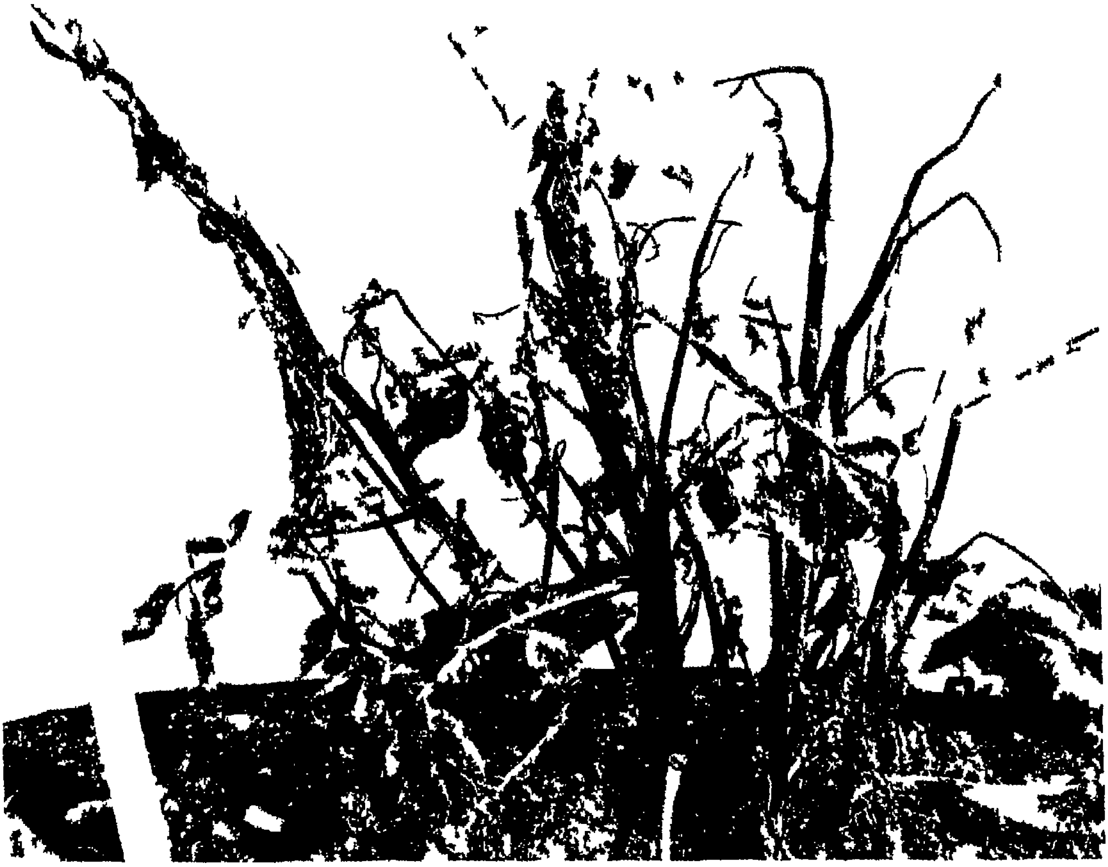

Fig. 2

ORTON SIRLAK DISEASE OF POT

# INFECTION EXPERIMENTS ON TOMATOES WITH PHYTOPHTHORA TERRESTRIA SHERB. AND A HOT WATER TREATMENT OF THE FRUIT

J. ROSENBAUM

Phytophthora terrestria Sherb. on tomatoes has been under observation in southern Florida for the past three years. During the early season of 1918–1919 it was hard to find a single fruit infected by this fungus in one of the tomato fields, while observations made in various parts of this same field later, following some exceptionally heavy rains, showed an infection of more than 66 per cent of the fruit, by actual count.

Sherbakoff,[1] referring to the rot caused by this fungus, states: "The rot occurs almost invariably only when the fruit touches the ground or is very close to it." In view of the high percentage of infection found in the field, it was supposed that if tomatoes rested or were placed on this infested soil, a high percentage of infection would result. A few experiments were performed, the results of which were somewhat contrary to what might have been expected. They give some additional information on infection, and suggest a possible treatment of the fruit for this disease in its very early stages.

## EXPERIMENT I

The purpose of the experiment was to determine whether contact with diseased soil is sufficient to produce a high percentage of infection.

Two hundred healthy, large green tomatoes were picked from a field known to have little if any Phytophthora, kept in the laboratory for two days so as to allow any infections present to develop, and then buried in an infested portion of the field in such a way that some of the tomatoes were only partially covered while others were two inches below the surface. There was no doubt that the land where the tomatoes were placed was infested, as counts made there a week earlier showed about 90 per cent to be infected.

The tomatoes were removed in small lots at twenty-four hour intervals, examined for any signs of infection, wrapped in paper and kept at room temperature, and again examined later. After a period of two weeks 14 per cent of the 200 fruits had become infected.

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[1] Sherbakoff, C. D. Buckeye Rot of Tomato Fruit. Phytopathology 7: 119–129. 1917.

The percentage of infection was much lower than was expected in view of the heavy natural infection at first found. The soil during the time of the experiment was relatively moist, but no rains occurred.

### EXPERIMENT II

The purpose of this experiment was to determine the influence of water on infection of the tomato fruit when in contact with diseased soil.

Flower pots were filled with infested soil obtained from the row where Experiment I was made, and one or more healthy, green tomatoes were placed in each pot and lightly covered with the soil. The pots were divided into three lots of twelve each. The treatments and the results obtained are summarized in the following table.

| LOT NUMBER | TREATMENT | PERCENTAGE OF INFECTION |
|---|---|---|
| 1 | Immersed in water for sixteen hours | 84 |
| 2 | Watered each day | 23 |
| 3 | Not watered | 0 |

The experiment was repeated in the laboratory and the results obtained were similar to the above. To determine whether the amount of water in the soil will influence infection in the field in a similar manner, two lots of ninety-six tomatoes each were buried in an infested field as outlined in Experiment I, in adjacent rows but separated by a soil ridge. One lot was watered each day. At the end of eight days both lots were removed. The lot watered had developed 63 per cent infection, while the adjacent lot had developed only 10 per cent.

These experiments leave little doubt that an abundance of water is necessary for an epidemic infection in the field. This is to be explained by the fact that moist infested soil containing mycelium of the fungus is not as infectious as infested soil where the zoospores are liberated and can swim about freely. Laboratory inoculations likewise have repeatedly shown that the zoospores can cause infection much more readily than any other stage of the fungus. The zoospore can penetrate and produce an infection on healthy, green fruit without previous injury.

### EXPERIMENT III

Working on the theory that the zoospores are liberated in the soil when an abundance of water is present, it was of interest to determine the effect of slight traces of copper in the water, when applied to infested soil.

One or more healthy tomatoes each were placed in infested soil in pots as for Experiment II. These were divided into several lots. The treatment and results are shown in the following table:

| LOT NUMBER | TREATMENT | PERCENTAGE OF INFECTION |
|---|---|---|
| 1 | Not treated | 3 |
| 2 | Watered heavily | 34 |
| 3 | Immersed in water for thirty-six hours | 55 |
| 4 | Immersed for thirty six hours in water containing 1–5000 $CuSO_4$ | 0 |

Stronger solutions of copper sulphate were used but no infection resulted in any case. A weaker solution than 1–5000 was not tried.

Immediately following the heavy rains referred to in the first paragraph, a number of growers picked their tomatoes. It was found that, notwithstanding the fact that most of these did not show any signs of infection at the time of picking, they developed the buckeye rot, due to *Phytophthora terrestria*, after being packed. In one case a grower was compelled to repack 500 crates on account of the rot, and less than one-third were found healthy.

One of the largest growers in southern Florida has made it a practice during the last three or four years to wash tomatoes in hot water before shipment. The method has given results that have led to the installation of such washing tanks in several packing houses along the East Coast. It was on this account that the following experiment with hot water treatment was made.

### EXPERIMENT IV

The purpose of the experiment was to determine the efficiency of hot water in preventing the spread of new and subcuticular infections of Phytophthora on tomato fruit.

In order to obtain fruit in the earliest stages of infection, a lot of green, healthy tomatoes were brought to the laboratory, washed, immersed in a 1–1000 mercuric chloride solution for five minutes, and placed in large moist chambers. They were inoculated in these chambers with a decoction of swarm spores, by means of an atomizer. Part of the inoculated fruit was removed approximately at the end of each twenty-four-hour period. Each lot of fruit as it was removed was divided into two parts, one of which was immediately immersed in hot water, while the other was left untreated. From previous preliminary experiments it had been found that green tomatoes can withstand water at a temperature of

60°C. for one and one-half minutes. If treated at 65°C. for the same length of time an occasional fruit will develop a rusty color and show slight shriveling, indicating injury from the hot water. Therefore, all the infected fruit was treated for one and one-half minutes at 60°C.

The following table shows the results obtained.

| NUMBER OF HOURS BETWEEN ORIGINAL INOCULATION AND REMOVAL OF FRUIT FROM CHAMBER | PERCENTAGE OF TREATED FRUIT DEVELOPING ROT | PERCENTAGE OF UNTREATED FRUIT DEVELOPING ROT | REMARKS |
|---|---|---|---|
| 24 | 0 | 100 | No infection was evident when fruit was removed |
| 46 | 0 | 100 | The infection was evident at time of treatment |
| 71 | 0 | 100 | As above |
| 96 | 66 | 100 | As above |
| 122 | 100 | 100 | As above |

The results of the experiment indicate that: (1) a period of *not more* (and possibly less) than twenty-four hours is necessary for the swarm spores to be in contact with the tomato fruit to produce infection; (2) immersing infected fruit in hot water at 60°C. for one and one-half minutes will prevent decay of infected fruit which does not yet show macroscopic signs of the lesion or those in which the tissues have not yet been penetrated deeply. In the experiment performed 71 hours after the inoculation was made the lesion was macroscopically evident; nevertheless, the hot water treatment stopped its further progress. Ninety-six hours after inoculation was made it was not possible to kill the fungus in the tissues in every case. One hundred and twenty-two hours after inoculation the invading mycelium had penetrated too deeply into the tissues to be affected by the hot water treatment.

The good results said to be obtained by some of the growers from treating fruit with hot water may be ascribed to the fact that not only are fungus spores and sand removed from the surface, but in addition it may also kill new infections which have not penetrated very deeply into the tissues of the fruit.

Repetition of all the experiments outlined here gave similar results.

## SUMMARY

An abundant moisture supply, preferably free water, in diseased soil, is a necessary condition for producing an epidemic infection with *Phytophthora terrestria* Sherb. on tomatoes. This is to be explained by the fact that only under such conditions will an abundance of zoospores be liberated.

The addition of 1–5000 copper sulphate solutions to infested soil will prevent infection of green tomatoes when in contact with such soil.

The immersion of green tomatoes in water at 60°C. for one and one-half minutes will prevent the spread of the buckeye rot where the infection is very recent and the fungus has not penetrated deeply into the tissues.

BUREAU OF PLANT INDUSTRY
WASHINGTON, D. C.

# BRIEFER ARTICLES

## PHONY PEACHES: A DISEASE OCCURRING IN MIDDLE GEORGIA[1]

DAVID C. NEAL

WITH PLATE IX AND ONE FIGURE IN THE TEXT

Phony peaches, apparently a new disease of the peach, has been known to exist for some time, especially in the peach belt of middle Georgia. In fact, the distribution of the disease appears to be more or less limited to that peach section of the state. This rather peculiar trouble of peach trees, although observed a few years ago by growers, as well as by Federal and State pathologists, has received practically no investigation, with the result that the factors concerned in its cause remain quite obscure.

The disease has been given the tentative name of "phony peaches" by growers, probably because of the more or less dwarfed character of trees which are affected. It seems appropriate to state that orchardists and pathologists have heretofore confused the phony disease and another somewhat similar trouble of the peach, namely, "collar-edema." The latter has been studied by Mr. M. B. Waite of the Office of Fruit Disease Investigations, Bureau of Plant Industry, and according to him the collar-edema disease of peaches is entirely distinct from the phony disease occurring in this state. Waite's[2] description of the collar-edema disease of peaches is as follows: "It is characterized by an edema or abnormal swelling of the collar of the tree, mostly below the ground line. The bark is thicker and fleshier or has a larger proportion of the flesh part than normal. The lenticels are enlarged and the wood is more or less brittle or corky, or less fibrous than normal. The trees are sometimes snapped off by the wind because of their brittle character. The growth is short and stubby and the leaves become yellow and drooped and usually fall prematurely." According to the same author, the collar-edema disease is restricted mainly to the eastern section of the United States, extending into New York State. It has also been observed in the State of Michigan. The phony disease of peach, however, has thus far been seen by the writer only in middle Georgia. With the exception of the short, stubby growth of the twigs, none of the above symptoms are evident on trees having the phony disease.

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[1] Published with the approval of the Georgia State Board of Entomology.

[2] From correspondence supplied the Georgia State Board of Entomology.

## DESCRIPTION OF THE DISEASE

### *Leaf symptoms*

A tree affected by the phony disease is readily detected, on account of its leaf symptoms. The leaves are somewhat larger than normal, and are especially marked by their dark green color. In typical cases this vivid green color of the foliage is so pronounced that such a tree is easily distinguished from normal ones by an observer passing an orchard, even though several paces away (plate IX, figs. 1 and 2).

### *Twig symptoms*

The twigs of trees having the phony disease are particularly noticeable because of their short, stubby growth. In cases where the current season's growth of normal shoots would average 2½ to 3 feet or more, a tree affected by the phony disease seldom averages one-third or one-half of this growth (fig. 1). As a result, the entire head of the tree is reduced to a rather compact shape, owing to the forshortened internodes and resulting close spacing of the leaves. With such malformations induced, the trees usually fall rapidly behind normal ones on the same soil as to growth and size.

### *Cause of the disease*

The present experimental work with this disease is so limited that the factors which are responsible for the symptoms which have been briefly described above are as yet unknown. The phony disease is possibly one of the so-called physiological diseases, falling perhaps in the same category with troubles like peach rosette, peach yellows, and the mosaic diseases of many plants. Since the disease is known to occur over a wide range of soil types, its relationship to such factors as soil fertility, soil moisture, malnutrition, or excessive physiological stimulation as the result of certain plant foods must all be studied under laboratory and field conditions before definite causes can be set forth.

### *Effect of the disease on fruit development*

Personal observations and conversations with growers who are familiar with the disease indicate that the amount of fruit which a phony tree sets is very small. Frequently fruit on such trees attains almost normal size, but the period of ripening is usually considerably retarded. There is some variation among the different varieties in this respect, but the fruit matures at least ten or twelve days later than normal, is insipid in taste, and has little, if any, market value.

### *The relation of age of trees to the development of the disease*

From field observations it appears that trees which have reached bearing age are more susceptible. Many cases are encountered among those which have attained four and five years, but the largest areas affected are found in orchards which are much older (five to ten years). The youngest case which we have on record is that of a tree which developed typical symptoms of the disease during its second season's growth.

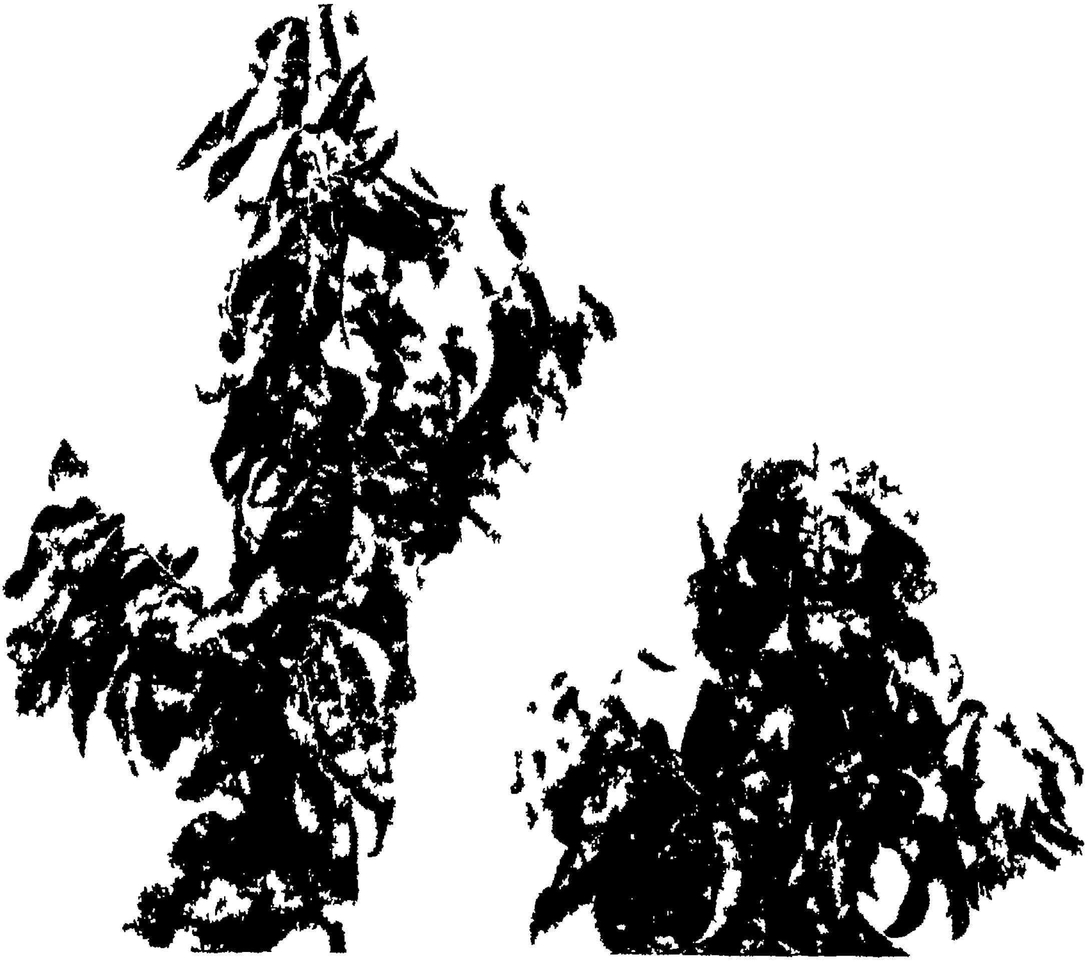

FIG. 1. COMPARISON OF THE CURRENT SEASON'S GROWTH OF NORMAL AND DISEASED SHOOTS

Healthy shoots at left, diseased shoot at right. Photographed August 16, 1919.

### *Varietal susceptibility*

The existence of varietal susceptibility or immunity does not seem to be established. In fact, most of the varieties of the peach which are grown in middle Georgia have been found to be affected to a certain degree. The disease is prevalent on Elberta, Hiley, Belle, Belle of Georgia, Mayflower, and J. H. Hale. It no doubt attacks other varieties. Owing to the frequent occurrence of the disease in plantings of Elberta, one is sometimes led to consider this variety more susceptible. On the other hand, it constitutes the largest number of trees of any single variety planted in this section, and this fact alone may account for the large number of such trees affected by the disease.

### *Presence of the disease on seedlings*

The phony disease has been found in the vicinity of Fort Valley, Georgia, on seedlings which are now in their sixth season's growth. Owing to this discovery, the nursery phase of the problem immediately presents itself for careful investigation. The following points appear to be extremely important in this connection, and are now being actively investigated by the writer. First, does the disease have its origin to any extent in the nursery, especially in the localities where nursery stocks are propagated for this section? Second, is the phony disease, like yellows and peach rosette, transmittable by budding?

Pending the outcome of experiments which are now in progress bearing upon this particular phase of the problem, it would appear to be only the part of sound judgment for the orchardist and the nurseryman to familiarize themselves with this relatively little known peach disease. They should certainly exercise the greatest caution in their selection of budwood from those regions which are known to be infested, in order to avoid disseminating the trouble to other localities which are free of the disease.

GEORGIA STATE BOARD OF ENTOMOLOGY
THOMASVILLE, GEORGIA

## PLATE IX

FIG. 1. A peach tree of the J. H. Hale variety now in its sixth year, showing typical symptoms of the phony disease. Note the short, stubby growth of branches and the dark green compact foliage. Photographed August 16, 1919.

FIG. 2. A healthy tree of the same variety and of the same age growing in an adjoining row. Photographed August 16, 1919

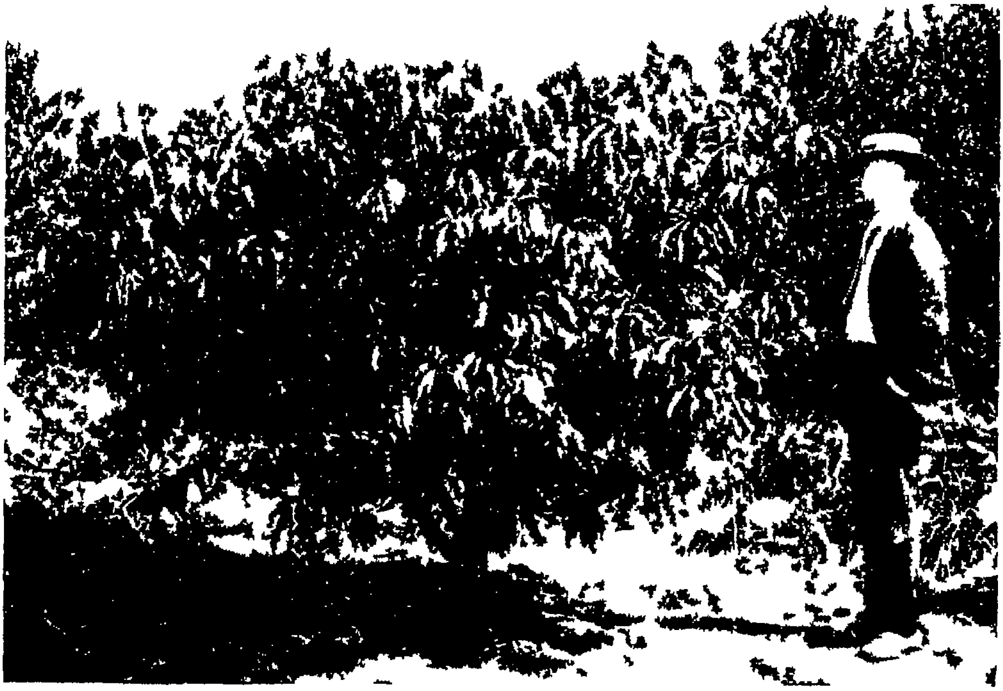

Fig 1

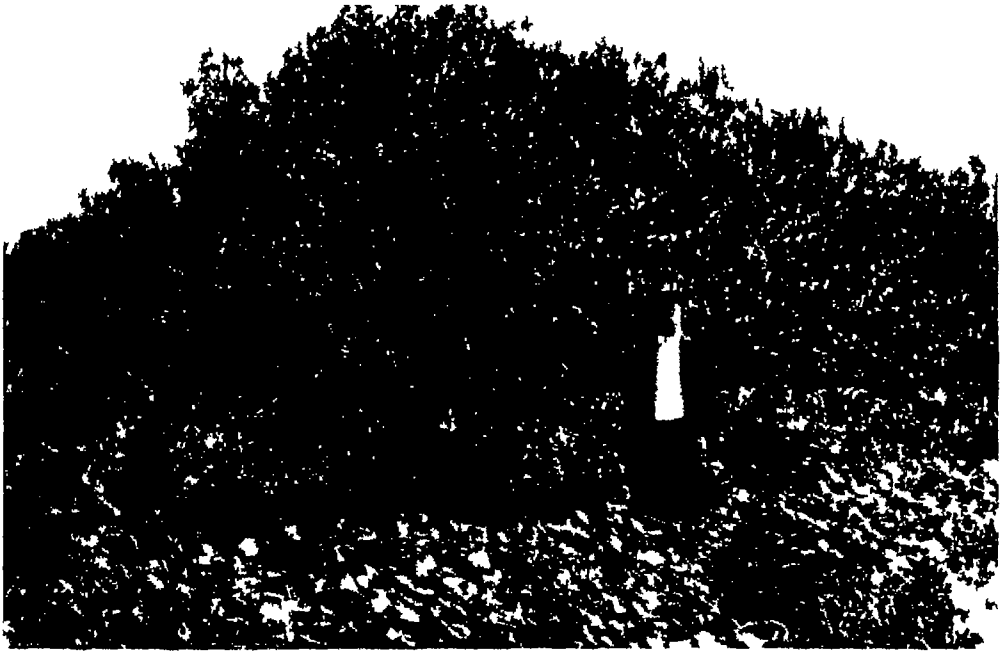

Fig 2

Neal Phony Peaches

## PHYTOPATHOLOGICAL NOTES

*Late seasonal production of aecia of Cronartium ribicola.* A freshly matured aecium of *Cronartium ribicola* was observed by the writer on a native tree of *Pinus strobus* near Amery, Wisconsin, September 16, 1919. It had developed at the upper end of a lesion, which had resulted from a natural infection and which had borne eight normal sized aecia early in the season. The lesion was on a branch which was about ½ inch in diameter at the place of infection.

The aecium was of recent origin since the newly ruptured peridium was filled with bright orange-colored spores. Dr. Perley Spaulding, of the Bureau of Plant Industry, Dr. S. B. Fracker and Mr. H. J. Ninman, of the Wisconsin State Entomologist's Office and Prof. John W. Stephens, of the University of Syracuse were with the writer at the time the aecium was found. Mr. Ninman stated at this time that one or two days previously he had seen two similar aecia in the same locality. The writer observed a newly developed aecium of *Cronartium ribicola* near Littleton, N. H., July 21, 1918. Aeciospores of this fungus are known to be carried many miles by the wind. Puzzling occurrences of this fungus on Ribes late in the season, which had been earlier apparently free from disease, may be explained by the presence of such late aecia. Judging from the observations of Miss Dosdall (Phytopathology **8**: 619, 1918) it seems that spores of such late seasonal development as mentioned above may over-winter in the aecia and become a source of early infection in the following season.

H. H. YORK.

*Personals.* Mr. H. F. Bain has been appointed pathological inspector for the Federal Horticultural Board, U. S. Department of Agriculture, to take charge of the treating of seeds and plants in connection with the inspection work at Washington, D. C. Mr. Bain is a graduate of Brown University and has recently received his discharge from the Army.

Dr. John P. Benson has resigned his position as assistant pathologist in the Office of Cotton, Truck, and Forage Crop Disease Investigations, Bureau of Plant Industry, to become assistant professor of agricultural extension in the University of California. He is now detailed as farm adviser to Fresno County, with headquarters at Fresno.

Mr. Chas. R. Hursh and Mr. A. A. Potter, formerly assistant pathologists in the Office of Cereal Investigations, Bureau of Plant Industry, have left the Departmental service, the former to take up graduate study at the University of Chicago and the latter to take up work in the agricultural division of the Bureau of the Census.

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COMPILED BY ALICE C. ATWOOD AND FLORENCE P. SMITH, BIBLIOGRAPHICAL ASSISTANTS, BUREAU OF PLANT INDUSTRY

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PHYTOPATHOLOGY

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# BACTERIAL BLIGHT OF SOYBEAN

FREDERICK A. WOLF

WITH FIVE FIGURES IN THE TEXT

## INTRODUCTION

For several years, especial attention has been given at the North Carolina Agricultural Experiment Station to the diseases of soybean, *Soja max* (L.) Piper. The most important diseases of this crop within the State have been found to be Fusarium blight or wilt, caused by *Fusarium tracheiphilum* E. F. S., and bacterial blight. Careful investigation of the former of these diseases has been made by Cromwell (2), whereas all existing published accounts of the latter have been found to consist of fragmentary notes.

The present studies on bacterial blight have been directed mainly toward the isolation of the causal organism, its identification, and the procurement of evidence of its pathogenicity. In addition, certain field observations and laboratory studies have been made during the past three seasons which further contribute to our knowledge of this disease and are consequently recorded in the present report.

## HISTORICAL ACCOUNT

The first mention of the occurrence of bacterial blight of soybeans appears to have been made by Heald (3, 4) in 1905. His account consists of a brief description of the appearance of the disease, with the suggestion that it is of bacterial origin. Subsequently, Smith (7) and Clinton (1) report the occurrence of a bacterial leafspot of soybean, and the latter author states that the disease in Connecticut is like the one described by Heald (3, 4) in Nebraska. Apparently, the only critical investigation of bacterial blight which has been made was conducted in Wisconsin by Johnson and Coerper (5). They isolated a white bacterial organism refer-able to the genus Pseudomonas, which they proved to be pathogenic. However, the detailed report of these isolation and inoculation studies and description of this organism appears never to have been made.[1]

A brief report of the present author's work in manuscript form, in which the causal organism is designated *Bacterium sojae* n. sp., was prepared by Tisdale (8). The manuscript on bacterial blight, dealing with the investigations conducted during 1917 and 1918, has been supplemented and modified by studies made during the past season and is presented herewith.

## APPEARANCE OF THE DISEASE

Bacterial blight of the soybean has been observed upon plants varying in age from seedlings to mature plants. It may appear upon the cotyledons or upon the true leaves in any stage of their development. Apparently stems and fruits are free from invasion by the parasite. The disease is, furthermore, widely distributed throughout the State, since it has been noted in practically every soybean field which has come under observation.

The first evidence of the disease which has been noticed is the presence of lesions on the cotyledons just as they are emerging from the ground. The lesions present at this time are usually marginal and are in the beginning little different in color from the normal tissues. They rather rapidly become brown, however, and meanwhile the tissues have shrunken and collapsed and the area of invasion may have enlarged, extending backward toward the hypocotyl. In extreme cases both cotyledons are destroyed and the hypocotyl is involved, thus causing the death of the seedling. Species of bacteria other than the parasite are always present in mature lesions on the cotyledons. In some instances one cotyledon only is involved in decay. Lesions which are marginal in origin are commonly irregular in outline, whereas those present on the outer surface of the cotyledons are commonly circular (fig. 3).

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[1] Since this manuscript was submitted for publication, a report of investigations on "Bacterial Blight of Soybean" (Coerper, Florence, M. Jour. Agr. Res. v. 18, No. 4, p. 179–194, pls. 12–18, one colored plate and one text figure, 1919), in Wisconsin, has appeared. This disease apparently differs in several respects from the one occurring in North Carolina in which, (1) the diseased areas are *typically* not so dark and usually remain intact; (2) the dried exudate is grayish and not brownish; (3) leaf parenchyma and cotyledons only are involved, since careful search during three seasons has failed to reveal lesions on petioles, stems, legumes, or seeds. The seed are believed to become contaminated by the washing of the parasite from the leaves only after the dehiscence of the legumes, and apparently normal seed, when planted, give rise to seedlings whose cotyledons are affected; (4) colonies of *Bacterium sojae* are smaller than *Bacterium glycineum* and the former organism is furthermore smaller than the latter. No strains of *B. sojae* with umbonate center, with wrinkled or convoluted surface, or which produce brown pigment, grow in the closed arm of fermentation tubes, or produce acid from the several sugars, have been isolated. At least a dozen strains have been isolated by the writer and three of them have been used, with concordant results, in the cultural studies.

The name Southern bacterial blight of soybean is therefore tentatively applied to the disease under consideration.

From the cotyledons the infection is spread to the first pair of true leaves and thence to other leaves. The occurrence of characteristic spots (fig. 4) which have manifestly arisen from infection from the cotyledons has often been noted on plants grown either in the field or in the greenhouse. These spots are first evident as small, water-soaked areas. The invaded tissues change in color to yellow, then to brown, becoming at maturity a very dark shade of brown or even purplish black. There is a retention of the translucent character, and individual lesions remain small and angular. The presence of a large number of lesions in close proximity may result in their confluence. Large, irregular dead areas may thus be formed, which may fall away, leaving perforations or notches. An indefinitely limited zone of yellowish tissue surrounds each lesion and may involve all of the intervening tissue (fig. 5). At this stage, dependent upon the severity of the infection, more or less defoliation results.

One of the distinctive features of the disease is the occurrence of a bacterial exudate on the lower leaf surface of the lesion. Under humid conditions, or if the leaves have been wet with rain or dew, turbid white droplets collect on the lesions. Otherwise, this exudate may appear as a hard, glistening film, which quickly redissolves when moisture is supplied.

## THE ORGANISM

### *Isolation*

A large number of isolations have been made from freshly invaded areas and mature lesions on leaves, from the exudate, from leaves which had overwintered in the field, and from lesions on cotyledons. Little difficulty was encountered in isolating the specific organism from any of these sources, especially after acquaintance with its appearance in culture. The organism was obtained often with an admixture of other species, but with the pathogenic form predominating, by one of the following methods:

1. The invaded areas, whose surface had been washed and disinfected in 95 per cent alcohol or 1:1000 mercuric bichlorid, were cut into pieces and laid, with aseptic precautions, upon the surface of nutrient agar plates. Abundant bacterial growth spread from the edges of these pieces of tissue. In a few cases the parasite could be transferred in pure culture

directly from these planted plates but most commonly the resultant growth was a mixture of species which were then separated by dilution cultures.

2. Small pieces cut from the lesions both directly and after surface disinfection were immersed in bouillon and after the bacteria had migrated from the tissues and diffused throughout the bouillon, poured plates were made. When proper dilutions were made isolated colonies of the parasitic form could thus be obtained in these poured plates.

3. A copious whitish exudate appeared on the lower leaf surface of areas which had been washed and disinfected. Some of this exudate was transferred by means of a sterile needle to tubes of sterile water, making a bacterial suspension from which successful platings could readily be obtained.

4. A lesion, with no previous attempt at surface disinfection, was thoroughly macerated in a drop of sterile water on a microscopic slide. A platinum loopful of this macerated material was transferred to one edge of a hardened agar plate and spread with the loop over the surface of the agar. Colonies of the causal organism, well isolated from other species, developed on these plates and were transferred directly to slant agar tubes. This is essentially the same method of isolation as is used in isolating *B. typhosus*. It is superior in the writer's hands to the other methods employed since it has yielded uniformly successful results.

## *Morphology*

*Vegetative cells.* The primary cause of soybean bacterial blight is a white, rod-shaped organism with rounded ends. When taken from lesions it is found to occur singly or in pairs. In nutrient broth, however, it may form short chains of four or five elements. The individual rods measure 1.2 to 2.6 by 0.8 to 0.9 μ.

*Endospores.* Endospores appear not to be formed, even in old agar cultures which were stained with Zeihl-Neelson carbol fuchsin.

*Flagella.* When the organism from lesions, from beef broth, and from agar is examined for motility, it is seen to possess the power of rather active locomotion. This movement is made possible by the presence of polar flagella, 3 to 5 μ in length, as has been demonstrated in four strains of the organism. Use has been made of Morrey's (6) modification of Loeffler's method on 18 to 24-hour agar cultures. Usually there is a single flagellum, although individuals with two, three, or four flagella have been observed (fig. 1b).

*Capsules.* A well-defined capsule is readily demonstrable by Welch's method (fig. 1c), whether the parasite is taken directly from invaded tissues or from culture on solid media.

*Zoogloeae.* Zoogloeae have not been seen in cultures on bouillon or milk. A thin pellicle appears at the surface of liquid media, but it never becomes zoogloeae-like.

*Involution forms.* No involution forms have been observed.

*Staining reactions.* The organism stains readily with carbol fuchsin (fig. 1a), methylene blue, and gentian violet, in both aqueous and alcoholic solutions. It is not acid-fast nor retentive of Gram's stain.

*Cultural characters.* In the cultural studies use has been made mostly of peptone-beef agar and bouillon to which either dextrose, lactose, saccharose, or glycerin had been added. The reaction, as indicated by phenolphthalein, following boiling of the solution tested, was neutral. The several carbon compounds were made up as 20 per cent solutions, sterilized, and only added with aseptic precautions to cooled agar or bouillon in the proportion of 5 cc. to 95 cc. of media. It was thought by this means to prevent the conversion of the complex carbohydrates through heating.

*Nutrient agar.* In agar poured plates incubated at 20 to 25°C. the colonies may appear within twenty-four hours or be first visible in forty-eight hours. At the end of four to five days the colonies are 2 to 3 mm. in diameter, circular, smooth, glistening, and possess an entire margin. They are white, raised, and soft, but not viscid in consistency (fig. 2). Buried colonies are lenticular.

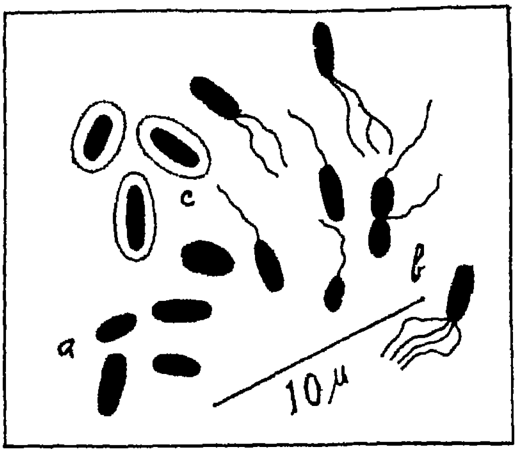

FIG. 1. *a*, *Bact. sojae* stained with carbol fuchsin; *b*, drawn from a preparation stained with Morrey's modification of Loeffler's method; *c*, the capsulate condition of *Bact. sojae* shown with Welch's method.

On agar slants there is an abundant filiform growth in forty-eight hours, which widens at the base of the slant. The streak is opaque, raised, and glistening, with a smooth or slightly contoured topography. No odor is produced and there seems to be no change in the color of the medium beneath the bacterial growth.

In stab cultures growth occurs only at the surface. After a month's time the surface growth had covered the greater portion of the tube.

*Gelatin stab.* The character of growth on gelatin stabs at 18°C. is confined to the surface. Within a month the surface will have been covered, but there is no evidence of liquefaction.

*Potato plugs.* Abundant growth occurs on potato cylinders, more spreading and flat than on agar, and grayish-white in color. The lower portion of the streak near the surface of the water is more glistening and more spreading than the upper part. No pronounced odor is developed and the substratum is not discolored. No change in medium is shown with iodine at the end of four weeks. A slight diastatic action in starch agar plates is evident, however, within two weeks.

*Bouillon.* Peptonized beef-extract bouillon becomes slightly clouded within forty-eight hours and there is little or no increase in cloudiness subsequently. A delicate pellicle is formed at the surface. No sediment is formed and no perceptible odor develops.

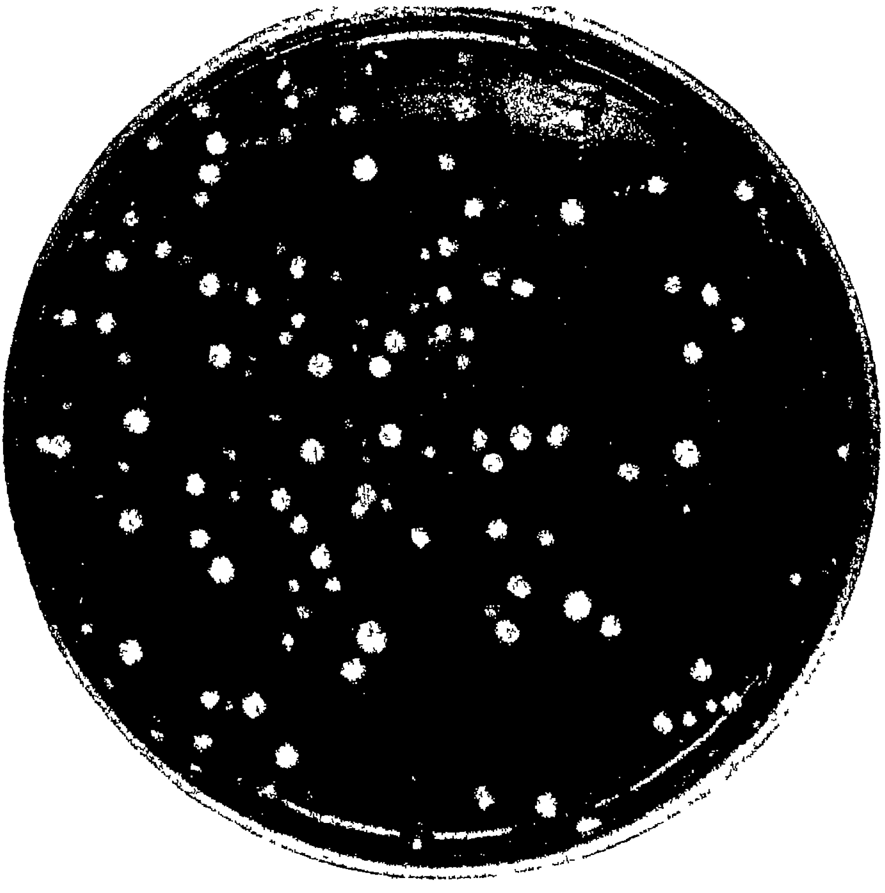

FIG. 2. POURED PLATE OF BACT. SOJAE ON NUTRIENT AGAR
The glistening surface with its lighter spots shows the reflection of windows

· *Litmus milk.* The litmus milk was made violaceous in color and subjected to sterilization in an Arnold sterilizer four times at intervals of twelve hours. A week after inoculation, the blue color is intensified and approximates caeruleus. After two weeks a white precipitate is apparent, which increases as growth proceeds. Within a month, the color becomes less intense as a slight reduction of the litmus takes place.

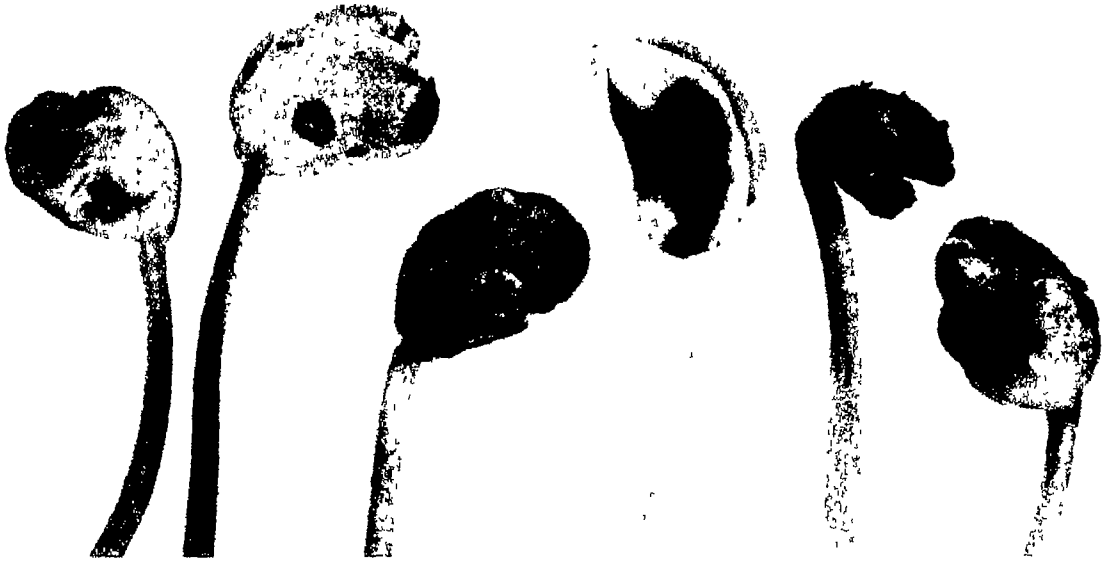

FIG 3. COTYLEDONS OF SOYBEAN SHOWING THE LESIONS PRODUCED BY BACT. SOJAE

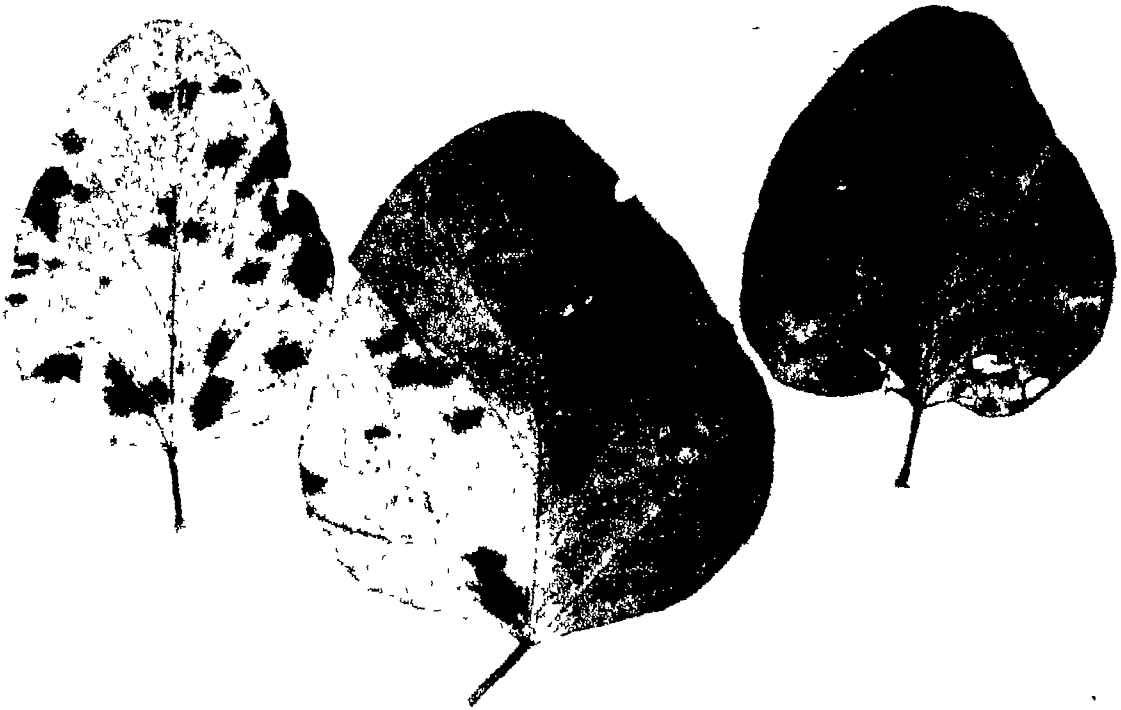

FIG. 4. FIRST TRUE LEAVES OF SOYBEAN, SHOWING CHARACTERISTIC LESIONS
Photograph taken with ray filter

### *Physical and biochemical characters*

*Litmus agar.* The carbon compounds, glycerin, saccharose, dextrose, and lactose from stock 20 per cent solutions, were added to cooled plain litmus agar tubes to make 1 per cent solutions of the respective sugars. Stab cultures on these media gave no evidence of acid nor gas formation after thirty days' incubation at 20°C.

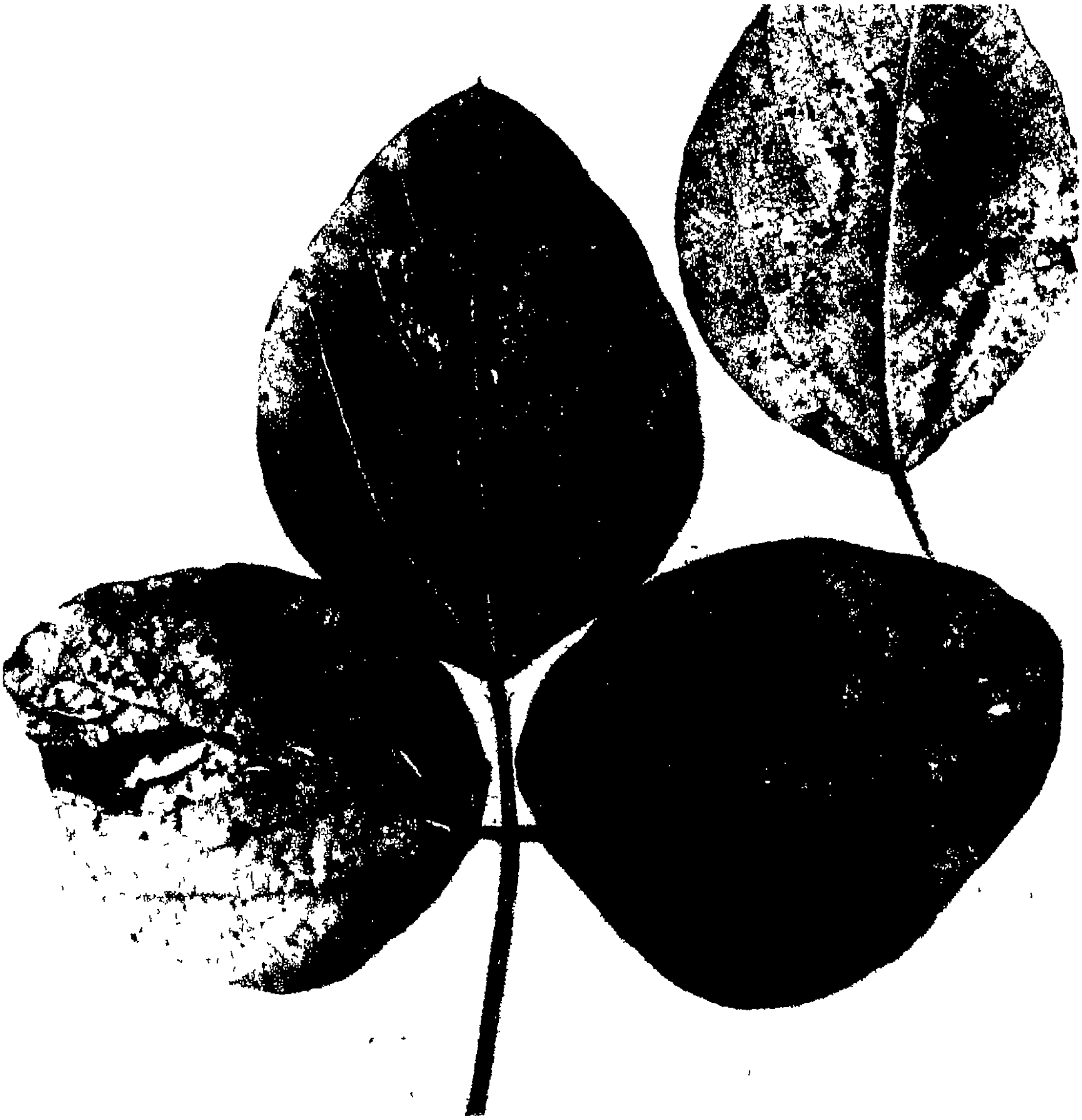

FIG. 5. LESIONS ON SOYBEAN LEAVES

Considerable chlorosis and fusion of lesions is apparent. Use was made of ray filter. One-half natural size.

*Fermentation tubes.* In peptone broth to which the several carbon compounds had been added no gas was formed in the closed arm of any of the fermentation tubes. There is furthermore no appreciable change in acidity. The media in the open arm becomes moderately turbid, with the most copious growth in solutions containing lactose. No growth takes place in the closed arm, indicating that the organism is strictly aerobic.

*Nitrates.* Tubes containing peptonized beef broth, to which 1 per cent potassium nitrate had been added, were used. At the end of five days, both control tubes and cultures were tested with starch-iodide solution. No blue color resulted indicating that there was no formation of nitrites.

*Indol.* Ten-day-old cultures in Dunham's solution were tested by using 0.01 per cent solution of potassium nitrite and a few drops of concentrated sulphuric acid. No pink color developed even on gently warming the tubes.

*Thermal death point.* The thermal death point of this organism as determined in the usual manner was found to be 52° C.

*Virulence.* The parasite can withstand considerable exposure, since it has been possible to isolate it from leaves which had overwintered in the field. No tests have been conducted to determine its sensitiveness in culture to desiccation, sunlight, and cold.

*Group number.* The group number, according to the descriptive chart of the Society of American Bacteriologists, is 212.3332023.

## COMPARISON OF THE SOYBEAN BLIGHT ORGANISM WITH OTHER BACTERIAL SPECIES ON LEGUMINOUS PLANTS

Several species of bacteria parasitic on leguminous plants have been described, from all of which the form on soybeans appear to differ. It is, therefore, given a new specific name, *Bacterium sojae*, from the host upon which it is parasitic. The specific differences of the several species are summarized in table 1.

### *Résumé of salient characters*

On the basis of the foregoing studies the parasite is manifestly distinct from species which are known to occur on leguminous hosts, and it may be briefly characterized as follows:

*Bacterium sojae* n. sp.[2] Rods cylindrical, rounded at ends, singly or in pairs; elements 1.2 to 2.6 by 0.8 to 0.9 $\mu$, motile by one to several unipolar

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[2] *Bacterium sojae* sp. nov., Baculis cylindracis, apicibus rotundatis, solitariis aut geminis, $1.2 - 2.6 \times 0.8 - 0.9\ \mu$, flagello uno polare aut pluribus flagellis moti-

TABLE 1

*Comparison of bacteria parasitic on Leguminoseae*

| ORGANISM | HOSTS | AVERAGE SIZE | GROUP NUMBER |
|---|---|---|---|
| Bacterium sojae n sp. | Soja max | 2 3 x 0 8 μ | Bact. 212 3332023 |
| Bacillus lathyrimanns and Taubenhaus | Lathyrus spp.<br>Trifolium spp.<br>Soja spp. | 1.4 x 0 75 μ | B. 211.2222522 |
| Pseudomonas pisi Sackett | Pisum sativum<br>Pisum sativum var. arvense | 2.2 x 0 68 μ | Ps. 211.2322033 |
| Pseudomonas medicaginis Sackett | Medicago sativa | 2 1 x 0 7 μ | Ps 212 3332133 |
| Pseudomonas phaseoli E. F. S. | Phaseolus vulgaris | 1 5 x 0 4 μ | Ps. 211 3322513 (?) |
| Bacillus leguminiperdus von Oven | Pisum sativum<br>Phaseolus vulgaris<br>Lupinus sp. | 2.1 x 0 8 μ | B. 111. . . . . (?) |
| Bacterium solanacearum E. F. S. | Arachis hypogoea<br>Stizolobium deeringianum<br>Phaseolus vulgaris | 1.5 x 0 5 μ | Bact. 21½.3333823 |

flagella, capsulate, no endospores, strictly aerobic. Gram-negative, not acid-fast.

Superficial colonies on beef-peptone agar, circular, raised, smooth, shining, white, with an entire margin.

Gelatin not liquefied; nitrates not reduced; neither acid nor gas produced in cultures with various sugars. Group number Bact. 212.3332023.

Pathogenic on leaves of *Soja max*, forming angular brown lesions.

Type specimens have been deposited in the herbarium of the Office of Mycological Collections, Bureau of Plant Industry, Washington, D. C.

libus; capsulatis; sporis non visis; aerobicis, methodo Grami non colorantis, cum acidibus decoloratis.

Colonis superficialibus in agar-agar orbicularibus, convexis, levibus, nitentibus, albidis; gelatinis non liquefacientibus. Nitrum non reducentibus. In mediis saccharatis neque gas neque acidum evolvatis.

Laesionibus foliorum Sojae macis primum translucentibus deinde brunneolis angularisque.

## INOCULATION EXPERIMENTS

The disease has been reproduced on soybeans with the characteristic symptoms, by artificial inoculation with pure cultures, in several series of experiments during 1918 and 1919. The original organism has been recovered in pure culture from five of these series and this reisolated organism has been used in four series of successful inoculations. Typical lesions followed the application of the organism to uninjured leaves. The inoculum consisted of a watery suspension of the organism and was applied by means of an atomizer.

The pure cultures used in the artificial inoculations were obtained from three sources, i.e., newly formed leaf lesions, lesions on the cotyledons, and from leaves which remained out of doors from the autumn of 1918 until March 10, 1919. Furthermore, a watery suspension has been made from the bacterial exudate taken directly from the lesions and this crude inoculum sprayed on healthy soybeans. Old leaves collected in the field during March were macerated with a mortar and pestle, covered with water for several hours and then the supernatant portion used as a crude inoculum by sprinkling it upon normal plants. Infections similar to the natural ones developed in all cases, both from pure cultures and from crude inocula. The plants used for inoculation were grown out of doors and in beds and flats in the greenhouse.

The following details will clarify the procedure employed in the inoculation experiments. A subculture was made on bouillon from a pure culture of the parasite. The cultures when twenty-four to forty-eight hours old were employed as the inoculum, after having been diluted with several volumes of water, application being made by means of an atomizer. The plants were first drenched by means of a watering pot and the inoculum was sprayed upon these wet leaves. Inoculations were made late in the afternoon and the plants were then covered with bell jars until the following morning. An incubation period of four to seven days followed. Mature lesions require two or three weeks longer for their development.

Several other leguminous plants, including cowpea, *Vigna sinensis*, velvet beans, *Stizolobium deeringianum*, bur clover, *Medicago denticulata*, and white clover, *Trifolium repens*, have been employed with negative results in artificial tests.

## HISTOLOGY OF DISEASED TISSUES

For the histological studies upon the pathology of bacterial blight, lesions in various stages of development, both from natural and artificial infection, were fixed in 95 per cent alcohol, embedded in paraffin, sectioned, and stained with carbol fuchsin or preferably alcoholic methylene blue.

Entrance of the parasite is very evidently gained through the stomates. In the young infections invasion was confined to the intercellular spaces, where the organism developed so abundantly as to completely occupy the spaces and to force out the air, thus accounting for the translucency of lesions. In mature lesions the intracellar spaces are rather closely packed with bacteria. Whether the collapse of the walls and the general invasion of older lesions is due to *Bact. sojae* is not known, since a yellow, one-flagellate organism is constantly associated with the parasite in old lesions.

## VARIETAL SUSCEPTIBILITY

Opportunity has been afforded, through the courtesy of the Department of Agronomy of the North Carolina Experiment Station, during the seasons of 1916 and 1917, to make observations[3] on all of the more common varieties of soybeans, including Mammoth Yellow, Haberlandt, Black Eyebrow, Tar Heel Black, Brown, Peking, Tokio, Virginia, Barchet, Chiquita, Guelph, and about 40 numbers from the Office of Seed and Plant Introduction. These varieties are grown in adjacent rows, giving ample opportunity for the spread of infection. Where evidence of difference in relative disease resistance did appear it is not believed to be due to innate qualities, although the observations are not sufficiently extended to justify passing final judgment.

## OVERWINTERING AND DISSEMINATION

No observations have been made on the extent of injury from bacteria blight to the seed, but it is believed to be of no economic importance It is believed, however, that the seed are primarily responsible for the overwintering of the disease and for its introduction into new localities. Seed infection does not appear to occur until after the pods have matured and opened since lesions upon the pods have never been observed. The wetting and drying of seed in the field would at least afford ample opportunity for the organism to be washed from the leaves and to gain entrance to the seed or adhere to its surface. As has already been pointed out, lesions may occur upon the cotyledons and may be the source of infection for the primary leaves. From these the disease is gradually spread to other leaves. At least a score of field observations throughout three growing seasons add indirect evidence bearing on the persistence of the organism in the seed. Fields in which soybeans are grown for the first time are commonly more or less affected with bacterial blight. In several instances these fields were miles from the nearest soybean field and were planted with seed from fields in which the disease is known to have been present during the previous year. Furthermore, plants coming from seed which has lain in infested fields during winter are early affected with blight. Since fallen soybean leaves decompose rather quickly, they are not believed to be responsible for infection at this time. The organism can hibernate, however, in leaves, as has been pointed out, in case the piling of the leaves prevents their decomposition.

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[3] From the notes of Dr. R. O. Cromwell, formerly Assistant Plant Pathologist, North Carolina Agricultural Experiment Station.

Water is beyond doubt the chief agent for local distribution of the parasite. Beating rains could readily scatter the bacterial exudate from the surface of invaded tissues to adjacent leaves. A series of observations in 1918, in a field in which soybeans had been planted in drills eight inches apart, showed convincingly the influence of showers upon the spread of the disease. In 1919, a field which had never previously been seeded to soybeans was found, on June 12, to contain an occasional plant whose primary leaves were badly diseased. A rainy period of several days' duration followed, and on July first, when next this field was visited, nearly every plant in the field bore numerous affected leaves. Attempts to associate the spread of soybean bacterial blight with leaf-hoppers, insects commonly present on soybeans, have given negative results.

## SUMMARY

Bacterial blight of soybeans is a disease whose presence appears to have been first recorded in Nebraska in 1905. It has subsequently been reported from Connecticut and Wisconsin, and the present investigation concerns its occurrence within North Carolina.

The lesions on the leaves begin as small, angular, water-soaked areas, which enlarge and change in color, becoming at length dark brown to purplish-black. The surrounding tissues are more or less chlorotic. Lesions may also be present on the cotyledons.

Under humid conditions a bacterial exudate may collect in whitish droplets on the lower leaf surface of lesions on soybean. This exudate on drying forms a film which covers the invaded area.

Infection appears to spread from the cotyledons to the true leaves and from these to other leaves.

The parasite has, on numerous occasions, been isolated from the invaded tissues and from the bacterial ooze. It is rod-shaped, flagellate, white in culture, is found to have the group number Bact. 212.3332023, and is herein described as *Bacterium sojae* n. sp.

When watery suspensions of the organism are applied to uninjured leaves infection will be evident within four to seven days.

Infection takes place through stomatal invasion, whence the organism passes into the intercellular spaces and probably becomes intracellular.

No evidences of differences in susceptibility of varieties of soybeans have been observed.

Infected seed are believed to be the chief means by which the disease is carried over winter and by which it is introduced into new localities. Leaves which remain out of doors over winter have been found to harbor this parasite. The disease is spread in the field by splashing rains.

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# SOME INSECT RELATIONS OF BACILLUS TRACHEIPHILUS ERW. SM.

FREDERICK V. RAND AND LILLIAN C. CASH

WITH ONE FIGURE IN THE TEXT

The studies on insect transmission of bacterial wilt of cucurbits by Rand[1] (1915) and by Rand and Enlows[2,3] (1916, 1920), after a temporary cessation due to the war, have again been resumed, and it has seemed advisable to set forth some of the results obtained during the past season.

## BEETLE DISSECTIONS AND INOCULATIONS

Previous tests had given infection[1] (1920) from the viscera, as well as somewhat less frequently from the mouth parts of a small percentage of striped and 12-spotted cucumber beetles (*Diabrotica vittata*, and *D. duodecempuncta*) fed directly upon wilted plants, when these beetle parts were directly inoculated into needle-pricked cucumber leaves. It was thought desirable to make similar tests throughout the summer using striped cucumber beetles taken from the fields at large. Collections began with the first spring appearance of the beetles in the vicinity of Washington, D. C. In no case were these insects brought into contact with the wilt organism (*Bacillus tracheiphilus*) subsequently to their collection in the fields. In cases of inoculation with the viscera alone the beetles were shaken three to five minutes in a 1–1000 solution of mercuric chloride in 75 per cent alcohol and then washed several times in tap water before dissection. Where the mouth parts also were used for inoculation they were first removed with a cold sterile scalpel, and the viscera then taken out under conditions precluding external contamination. Inoculations were then made by macerating the part to be tested in a drop of water and pricking it into a young cucumber leaf. In no case were the plants placed in damp chambers but usually the inoculations were made under shade. In all cases of positive results the perfectly definite signs of the disease were further checked up by tracing the origin of wilt to the inoculation points and by a microscopical examination of the cut stems showing the vessels to be occluded with bacteria. In the first two cases *Bacillus tracheiphilus* was plated out and successfully inoculated into cucumber plants. All inoculations up to May 28 were made upon potted cucumber plants in the greenhouse at Washington, D. C., and subsequent inoculations were made at Tuxedo, Md., inside of large field cages covered with wire netting 18 meshes to the inch. The details of this series of dissection inoculations follows:

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[1] Rand, Frederick V. Dissemination of bacterial wilt of cucurbits. Jour. Agr. Research 5: 257–260, 1 pl. Nov. 8, 1915.

[2] Rand, Frederick V., and Enlows, Ella M. A. Transmission and control of bacterial wilt of cucurbits. Jour. Agr. Research 6: 417–434, 3 fig., 2 pl. June 12, 1916.

[3] Rand, Frederick V., and Enlows, Ella M. A. Further studies of bacterial wilt of cucurbits. U. S. Dept. Agr. Bul. 828. 1920.

*May 19*: 165 male and 90 female beetles were collected from the earliest cucurbit (cymbling or scallop squash) field in the vicinity of Tuxedo, Md.; dissected and inoculated the following day. Wilt resulted from the viscera of one male and one female beetle, and *Bacillus tracheiphilus* was isolated from each of these plants and successfully inoculated again into cucumber plants These beetles were collected fifteen to eighteen days before wilt appeared in this field or in the vicinity of Washington.

*May 22:* 197 males and 99 females from the same cymbling field; dissected and inoculated the following day. Wilt resulted from the viscera of one male.

*May 24·* 96 males and 55 females from Giesboro Point, D. C.; dissected and inoculated three days later. Results negative.

*May 27.* 25 males and 24 females from the same locality; dissected and inoculated the following day. One case of typical wilt started from the visceral inoculation of one male beetle but the plant was accidentally destroyed before final notes were taken.

Collections of May 28 to June 6 from cucumber and cymbling fields in the vicinity of Tuxedo, Md., and Giesboro Point, D. C., aggregating 297 males and 192 females; dissected partly on the date of collection, partly on the day following and in a few cases on the second day following, all gave negative results.

*June 11:* 22 males and 6 females; dissection and inoculation at once from mouth parts and viscera separately. Wilt resulted in inoculations from the mouth parts of one male and one female respectively, but in no other cases of mouth parts or viscera. These beetles were collected from two cymbling fields where an occasional natural case of wilt had developed subsequently to June 6, and about half of the beetles were taken directly from wilting plants.

*June 16·* 60 males and 23 females collected on healthy young cucumber plants at Greenport, Long Island, N. Y., in a field where an occasional wilting plant was to be found. These beetles were fed for five days on young bean plants (non-susceptible to wilt) before dissection and inoculation at Tuxedo, Md. The viscera of two males and one female gave typical wilt infection.

*June: 18:* 32 males and 7 females collected on cymbling in the vicinity of West Plains, Long Island, N. Y.; dissected and inoculated three days later at Tuxedo, Md. Results negative. No wilt was present in the field where those beetles were collected.

*June 18:* 70 males and 14 females from young cucumbers near Calverton, Long Island, N. Y.; dissected and inoculated 3 days later at Tuxedo, Md. Wilt resulted from the viscera of one female. No wilt was found in this cucumber field nor in the vicinity of Calverton. In fact, most of the cucurbits in this locality were just breaking through the ground.

*June 21:* 63 males and 38 females from the cucumber variety plats, Tuxedo, Md.; dissected and inoculated at once from mouth parts and viscera separately. Infection followed from the viscera of one male, but none from the mouth parts of any.

*August 6:* 50 beetles, in part recently pupated adults from the experimental plats, Tuxedo, Md.; dissected and inoculated on the same date. Wilt resulted from the viscera of one mature female.

*August 18:* 48 beetles in part recently pupated adults from the same field, dissected and inoculated on the same date. Results were negative.

Some of these positive results were obtained from the first beetles collected (May 19) in the earliest cucurbit plantings in one locality, whereas careful observations in this locality revealed no cases of wilt until eighteen days later (June 6), these few cases having developed in the experimental plats between the observations of June 3 and June 6. Including all cases where the sex of the beetles was recorded (up to August collections) an aggregate of 1027 males and 548 females was used. Of all positive results from visceral inoculations with these beetles 6 were from male and 3 from female individuals. Since approximately twice as many males as females were used the ratio between male and female carriers was therefore one to one.

One of the visceral infections followed from inoculation on the date of collection; four from inoculation one day after collection, one from inoculation three days, and three from inoculation five days after collection. In the latter case the beetles were fed during the interim upon young bean plants, in all other cases upon young, healthy cucumber seedlings free from beetle injuries. The greater portion of the inoculations were made one day after collection of the beetles, and with the exception of an occasional 12-spotted beetle all were striped cucumber beetles.

Out of the two sets of inoculations with mouth parts of beetles taken in part from wilting plants in the field, infection resulted from one male and one female out of an aggregate of 85 males and 44 females.

### SUPPLEMENTARY EXPERIMENTS

As a check upon these dissection and inoculation tests a portion of each collection of beetles (several hundred in all) made between May 19 and 29 were enclosed with young cucumber plants in a new field cage about 40 feet square, covered with new 18-mesh wire netting and provided with a solid board base extending 18 inches into a clay subsoil. In this cage on June 11 several cases of typical wilt were found starting from beetle injuries on the leaves. On July 3 the plants in this cage were carefully gone over and 19 cases of typical wilt found—all having apparently started from beetle injuries and all unquestionably from the beetles inserted. Pure cultures of *Bacillus tracheiphilus* were obtained from several of these plants and subsequent inoculations with these cultures were successful. It should be recalled that all of these beetles were collected and imprisoned before there was any wilt in the surrounding fields.

As a corollary to this experiment about two dozen small cone cages of 18-mesh wire netting were placed over as many young cucumber hills (June 4), the striped cucumber beetles present and gnawing the leaves first being driven away. One case of wilt, the first of the season in the vicinity of Tuxedo, Md., was observed under one of these cones on June 6, the first case in the field. On June 11 five other plants under five other cones were found wilting. All these cases had started from injuries made by cucumber beetles before the application of the cones on June 4, and no beetles had been found on any of these plants since that date. Wilt first appeared in the cucumber variety field outside of the cages on June 6 when about a dozen cases were found, all likewise having started from beetle injuries.

In all cases of natural infection seen during this series of studies (1914–1920) in which observations were made early enough in the course of the disease to determine the point of origin, the wilt clearly has always started from injuries identical with gnawings known to have been made by cucumber beetles. Experiments already reported, loc. cit. 2, 3 (1916, 1920), have shown that stomatal infections do not take place. In addition to these results, recent experiments seem to indicate that infection does not take place unless the bacteria are brought into contact with injured portions of the vascular system. In several series of experiments with young cucumber plants the epidermal and subepidermal cells were scratched with a sterile needle without going deep enough into the tissue to reach the vascular bundles. The surface of these scratched stems were smeared with virulent wilt bacteria. In no case did wilt follow although 100 per cent infection resulted when these same cultures were smeared over the deeply pricked stems or leaves of similar plants.

The results of these tests, as of the early spring dissection and inoculation experiments, indicate that a small percentage only of striped cucumber beetles harbor the wilt organism when or soon after they enter the cucurbit fields in the spring, and that the bacteria thus carried gain entrance only through fresh injuries reaching to the vascular tissues.

## INTESTINAL ISOLATIONS

The dissection and inoculation tests of this and preceding seasons have demonstrated the presence of *Bacillus tracheiphilus* in the intestines of occasional striped and 12-spotted cucumber beetles. It only remained to actually culture out the organism. On November 8, 1919, twelve striped cucumber beetles previously fed on wilting cucumber plants for two weeks were held in a clean glass jar without food for four hours. These beetles were then placed in a sterile test tube and shaken for a moment in 95 per cent alcohol and then for two minutes in mercuric chloride water (1–1000). The beetles were then washed four times in sterile tap water, placed in separate sterile Petri dishes and the viscera removed by cutting the tip of the abdomen with a sterile scalpel and squeezing out the contents onto the sterile glass by an oblique backward movement of the scalpel. The viscera from each beetle were macerated in a test tube of sterile beef bouillon, let stand for half an hour and then dilutions made in further tubes of bouillon, whereupon Petri dish cultures were made in plus 15 beef agar. In this manner *Bacillus tracheiphilus* was obtained in pure culture from the viscera of three (Nos. G, I, K) out of the twelve beetles tested. These cultures were checked up by successful inoculations into young cucumber plants.

The abdominal remains of each of these twelve beetles were macerated in a drop of water and used at once to inoculate the pricked leaves of twelve young cucumber plants, as a check on the isolation test. Typical wilt resulted from one (No. K) of these direct inoculations, and this was one of the three beetles giving the wilt organism from the visceral isolations. The juice of this wilted plant was used to directly inoculate a healthy cucumber plant which later developed the disease.

## FECAL INOCULATIONS

Field observations in the early morning had many times shown what appeared to be the feces of cucumber beetles mixed in the dew over portions of cucumber leaves showing beetle injuries. Subsequently some of these leaves were found with bacterial wilt starting from the beetle injuries. Furthermore, preliminary field experiments in which different sets of caged cucumber plants were artificially inoculated by needle pricks from pure culture (1) after sundown and (2) on the following morning when it was hot and sunny, had given a higher percentage of infection from the evening inoculations. In one test all of the six plants inoculated at nightfall developed wilt, while all of the six plants inoculated the following morning remained free from the disease. These facts suggested the possibility of fecal infection in the dew over fresh abrasions on the leaves.

Accordingly a series of experiments was carried out to clear up this point. On November 8, 1919, 18 striped cucumber beetles, previously fed for three weeks on wilting cucumber plants, were placed in a clean, dry glass vial and the feces collected after two hours. This fecal matter was then macerated in a few drops of water and inoculated by needle pricks into the leaf of a young healthy cucumber plant in the greenhouse. The results of this experiment were negative. This test was repeated, collecting the feces over night, and with a like negative result.

As a result of these two experiments it was thought possible that if the feces were not allowed to dry, and if air conditions comparable to the dew point in the field were given, a different result might follow. Accordingly on December 25, 16 striped cucumber beetles, previously fed for seventeen days on wilting cucumber plants, were caged in small vials, 4 to a vial, directly over young cucumber leaves in such a way that the feces would drop through to the leaves without allowing the beetles themselves to come into contact with the leaves (fig. 1). The cucumber leaf was supported upon a heavy cork and covered with another piece of cork ⅛ inch thick. A section of this thin cork under the bottomless vial was cut out and the lower end of the vial separated from this opening through the cork by a small piece of galvanized iron wire netting, 18 meshes to the inch. The leaf was moistened, pricked with a sterile needle, and the beetles were then introduced through the upper opening of the vial, which was closed with a cork stopper and the whole apparatus held in a damp chamber over night. The presence of the cucumber leaf and the access to air below, kept the beetles most of the time near this end of the vial so that the feces readily fell through the netting and the following morning were observed on the leaf below. The beetles themselves, however, could in no way reach the leaf. The apparatus was set up at night and removed the following morning, after which the plants were held in the beetle-free greenhouse for observation. On December 6 one of the four plants was starting to wilt from the inoculated portion and three days later the whole plant had succumbed. Microscopical examination of the cut stem showed the presence of bacteria in the vessels and direct inoculation of a healthy cucumber plant with the juice from this stem induced the disease.

These same beetles were then caged for three days more on a wilting cucumber plant and the experiment repeated in an identical manner, and with the same results. One plant out of the four wilted and *Bacillus tracheiphilus* was isolated from it and subsequently checked up by successful inoculation into a healthy cucumber plant.

As a further control on this series of experiments the same set of beetles was caged over night with a wilted plant and then (December 29) removed and each of the sixteen beetles at once dissected. Inoculations were made to separate plants with the mouth parts and viscera and all held over night in a damp chamber. Four cases of wilt resulted from inoculations with the mouth parts of the sixteen beetles and five cases from those inoculated with the viscera. Two of the mouth part infections were from beetles showing no visceral infection.

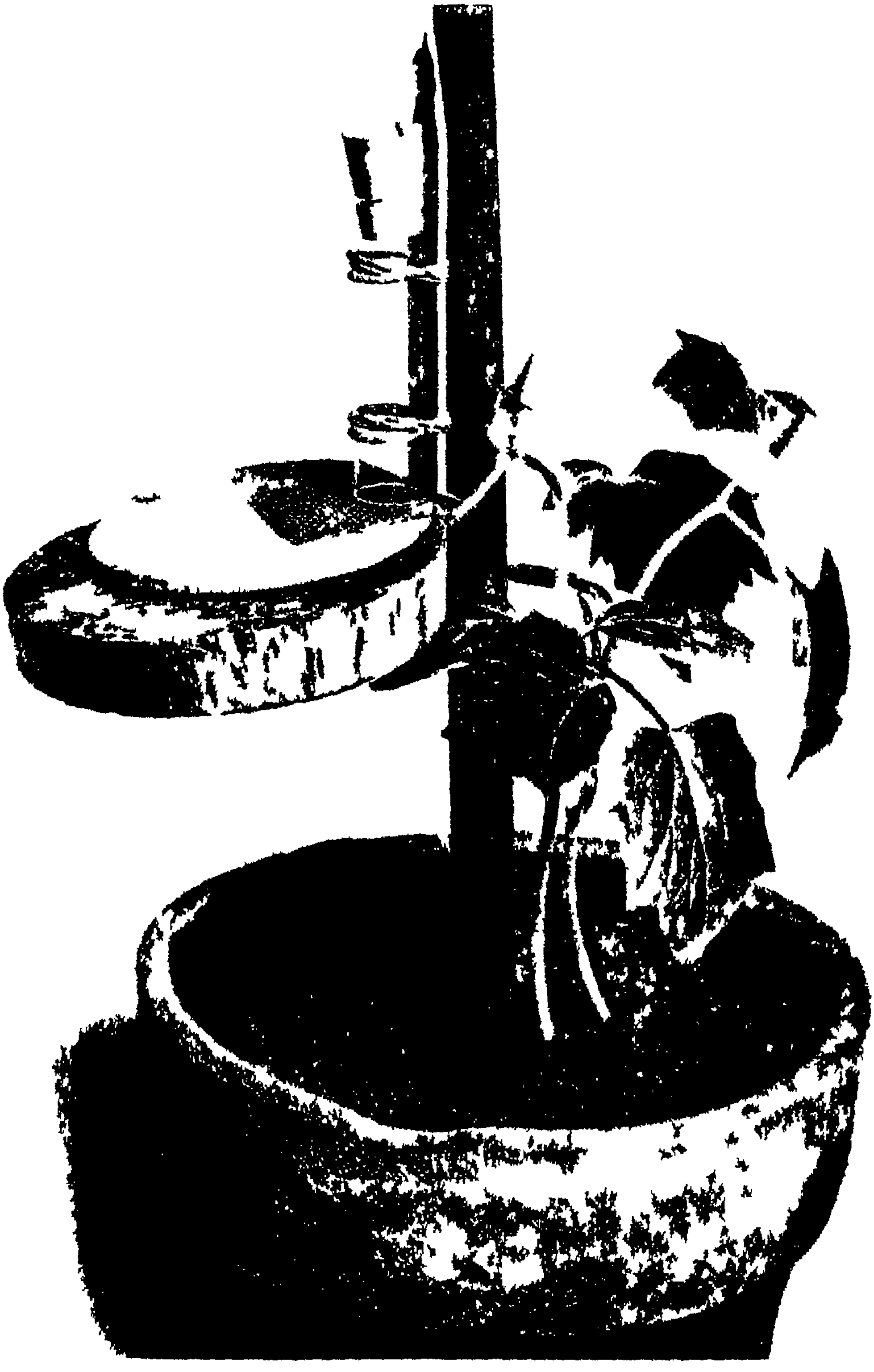

FIG 1 APPARATUS USED FOR OBTAINING FECAL INFECTIONS WITH BACILLUS TRACHEIPHILUS WITHOUT CONTACT BETWEEN INSECTS AND PLANT
Photograph by J F Brewer

In a subsidiary test (December 6) where seven beetles, previously fed for eighteen days on wilting cucumber plants, were taken directly for inoculation, two cases of wilt resulted from the visceral inoculations but none from the mouth parts. These plants were not held over night in a damp chamber.

## SUMMARY

It is clear that a small percentage of striped cucumber beetles harbor the wilt organism internally when they enter the cucurbit fields in the spring and there appears to be no other source for their infection than the cucurbit crop of the preceding autumn. Infections may take place from the mouth parts, at least for a limited time after feeding upon wilted plants, and infections are shown to occur when the feces of some beetles, but not all, come into contact with fresh injuries to the leaves. Infections take place only through injuries involving the vascular system. *Bacillus tracheiphilus* has been isolated directly from the viscera of wilt-fed striped cucumber beetles. Beetles of the genus Diabrotica (proved for two species) are the only carriers of the disease so far known in nature.

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# NOTES ON SOME DISEASES OF ASPEN

Carl Hartley and Glenn G. Hahn

With Three Figures in the Text

The quaking aspen, *Populus tremuloides* Michx., probably the most widely distributed American forest tree, is in places unusually subject to disease. In the Pike's Peak region in east-central Colorado diseases are so destructive as to suggest that they may be quite as largely responsible there for the short life of aspen stands as the shading by the more tolerant conifers, to which aspen replacement in the East is commonly ascribed. The damage done by disease in the aspen in this district is not equalled in any other native American tree of which the writers have knowledge in any part of the country. Death of isolated trees as well as of those in groups regularly occurs much before maturity. Observations in the Wasatch Mountains of Utah, where aspen seems to be a less temporary forest type, indicate that diseases are decidedly less destructive there. The most prominent of the diseases noted are described in the following:

## LEAF TROUBLES

The most prevalent leaf disease in the Pike's Peak region is that caused by *Sclerotium bifrons*. The disease is characterized by the death of the entire leaf. Half of the foliage over considerable forest areas is sometimes killed. The greatest prevalence is near streams. The dead leaves are typically persistent until autumn. In August one to five, or more, definitely outlined areas in each leaf become thicker and darker than the rest of the leaf, and usually drop out before the leaf falls, resulting in a shot-hole effect. The sclerotia vary from brown to black, with nearly white interior tissue. They are circular, ellipsoid, or sometimes irregularly lobed, with diameters usually between 2 and 8 mm., 3.5 mm. being a very common diameter. Attempts were made to develop fruits from these sclerotia. Fresh sclerotia were washed in mercuric chloride solution and planted in six different kinds of nutrient agar (prune, rice-stem, potato, cornmeal, beef and saccharose, and cornmeal and saccharose). The cultures either remained sterile, or yielded only *Cytospora* sp., which fruits commonly on dead aspen bark in the Pike's Peak region. Sclerotia placed on the ground in loose cloth sacks near the point of collection (9000 feet elevation, Pike National Forest) and at Great Falls, Virginia, in the autumn, showed no change by the following spring. Possibly cultures from these over-wintered sclerotia would have been more successful.

The fungus has been found abundantly by Dr. G. G. Hedgcock and the writers on the Pike, Colorado, Rio Grande, San Isabel, San Juan, Uncompaghre, and Montezuma National Forests, Colorado, and by Dr. Perley Spaulding practically throughout New England and the Adirondack region. In Utah it has been observed, but less commonly. Specimens have been secured from the Hayden National Forest, Wyoming, collected by Mr. A. M. Cook. In the Adirondacks, Dr. Spaulding's notes state that in mixed stands of *P. tremuloides* and *P. grandidentata* only the former is attacked. The fungus has also been reported from Wisconsin (2). Specimens have been distributed as N. A. F. 2554 (from Ontario) and Fungi Columbiana 155 (Ottawa) and 2276 (Colorado). Specimens are now in the collections of the office of Forest Pathology, collected by Dr. G. G. Hedgcock, Doctor Spaulding, Mr. G. L. Barrus, and the writers, from *P. tremuloides* in Colorado (fifteen collections), New York (three), and Vermont (two). The only collection known to the writers on hosts other than *P. tremuloides* is from Lombardy poplar (*P. nigra italica* Du

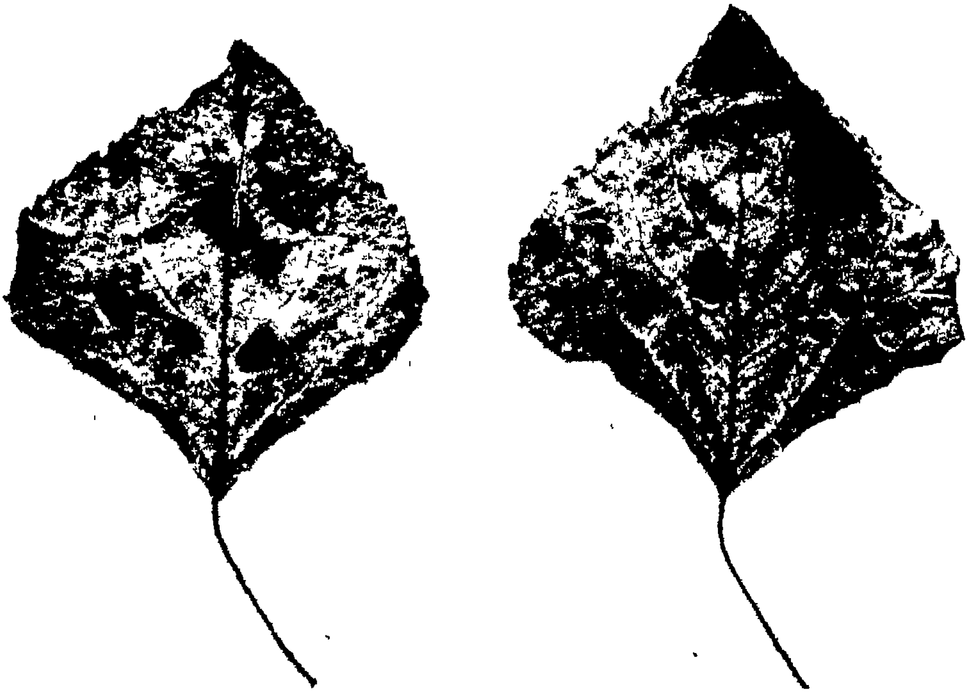


FIG. 1. LEAVES OF LOMBARDY POPLAR KILLED BY SCLEROTIUM BIFRONS

The sclerotium in the right-hand leaf has commenced the process of separation from the leaf which ultimately gives a shot-hole effect.

Roi), received from Miss Ethel Paine, Heath, Massachusetts (fig. 1), (Specimen F. P. 29306).

In the spring or early summer both the youngest leaves and the tender twig tips are sometimes killed in such a way as to suggest frost injury but at times when there has been little frost While the leaves do not persist and bear sclerotia, there is reason to think that this phenomenon is also due to the Sclerotium.

Cultures made from an angular leaf spot rather prevalent on the Pike National Forest yielded, in additon to hyphomycetes, nothing but *Cytospora* sp.

Leaf rust, *Melampsora albertensis* Arth. (Specimens F. P. 32698), is also quite common in the Pike's Peak region, fruiting conspicuously on the lower surfaces of the leaves, but does apparently little damage, not causing the premature defoliation often observed in young *Populus deltoides* affected with *Melampsora medusae* Thüm., or the blighting of leaves and lateral twigs by *Marsonia populi* (Lib.) Sacc. (5). This blight has been observed by Dr. G. G. Hedgcock to be frequent and injurious upon the Holy Cross National Forest, Colorado, and Targhee National Forest, Idaho.

## TWIG TROUBLES

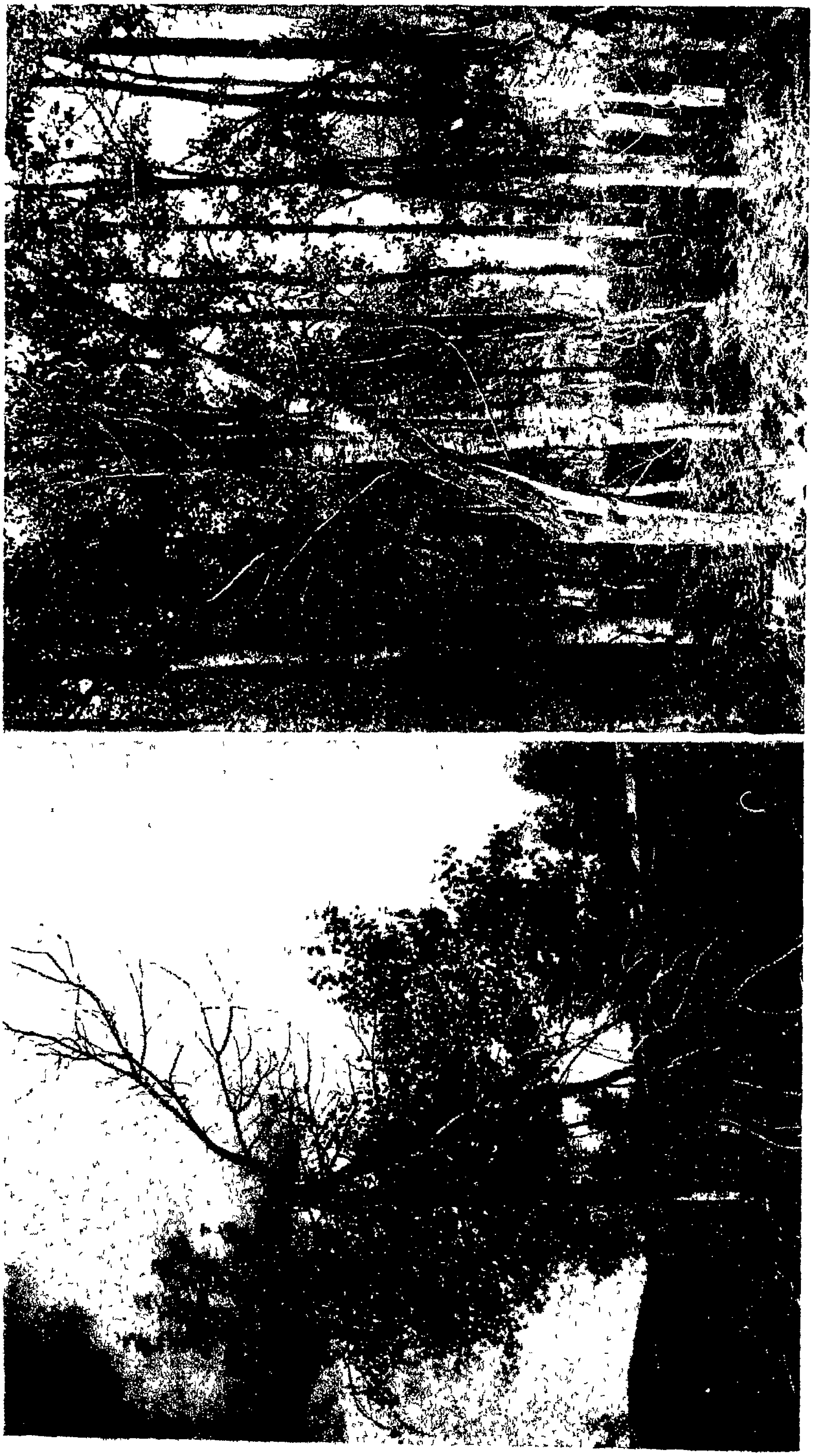
A disease locally prevalent in the Pike Forest, which has been observed in late summer during several different seasons, is a twig blight. The persistent blackened leaves give the trees much the same appearance as pear trees affected by fire blight. Insect tunnels were found in some of the affected twigs.

A trouble which may or may not have been connected with the death of twig terminals was a killing of short lateral twigs and isolated leaves with the same characteristic blackening and persistence. Examination of solitary dead leaves showed in part of the cases an insect larva tunnelling in the twig directly under the point of attachment of the petiole.

## TRUNK TROUBLES

The heart rot due to *Fomes igniarius*, which is widely distributed on aspen (7), is common in parts of Colorado, entering relatively small stems and, after establishment in the trunk, spreading outward through both heart and sap wood until the cambium is reached and killed. Ordinarily however only the heart wood and older changing sap wood are affected.

The most serious damage to the aspen in the Pike's Peak region is from bark lesions. Three types are frequent: (1) Elongated dead areas most often formed on the south or west sides of trunks, presumably due to


2 3

FIGS. 2 AND 3. POPULUS TREMULOIDES AFFECTED WITH TYPE II CANKER, BROKEN OVER AT THE REGION OF THE LESION

Photographed by Dr. Perley Spaulding, Saranac Inn, N. Y. 1909 winter sunscald. These sometimes serve as starting points for lesions such as described under III, but do not directly cause death. They have not been commonly observed. II. Determinate cankers which may appear on any side of the tree and are not believed to be connected with sun or winter injury, though such a possibility is not excluded. In size this type of canker is usually in the earlier stages small, and may become exceedingly large, lengths of 12 to 15 feet having been observed, at times practically involving the larger part of the tree In appearance these suggest cankers caused by *Bacillus amylovorus* (Burrill) De Toni. Observation indicates that some of these cankers continue to spread, successive zones of wound callus being killed. In certain cases the parasite seemed to be able to extend the lesion the second year only at the upper margin. Lesions on the trunk often originate at branch stubs. Branches as well as main trunks may be affected. The type II canker is the most serious in the Pike's Peak region as well as in the East throughout the New England and Adirondack regions. In the latter region this canker renders the aspen worthless before it reaches available size. Here the canker, according to the observations of Dr. Spaulding, causes the aspen to be readily susceptible to mechanical injury, trees frequently breaking over at the region of the lesion (figs. 2 and 3). III. Lesions on less vigorous trees, which spread more rapidly and are not often determinate or regularly zonate. These frequently cause death by girdling. All three of these types of stem lesions are common in slow-growing stands on unfavorable sites. As these sites are in general dry, it seems probable that it is the lack of vigor of the host more than favorable conditions for parasite development which is responsible for the local variations observed in prevalance of cankers. A *Cytospora* sp. with reddish or orange-colored spore horns and closely resembling *Cytospora chrysosperma* Pers. fruits abundantly on cankers of types I and III, and very promptly on aspen killed back by any agency. On cankers of type II it seems less common than on I and III. *Dothiorella populnea* Thüm. (Specimen F. P. 32699) is frequently found mixed with the Cytospora, the pycnidia crowded on a stroma, erumpent, and fully exposed when the epidermis is entirely loosened and falls away. This fungus is of interest for it is listed by Stevens (8) as being perhaps parasitic on Populus. *Valsa sordida*[1] has also been collected on the cankers.

An interesting phenomenon possibly related to a bark disease of established trees is the bark blackening of cuttings. While *Populus alba* (4) and *P. tremula* (1) cannot be propaged from cuttings, Hedrick (6) states that cuttage is a practicable method for propagating *P. tremuloides* In an effort to test the value of aspen as a nurse tree, Mr. C. G. Bates, of the Forest Service, in 1909 set out at Halsey, Nebraska, 2600 hardwood aspen cuttings, part from South Dakota, part from an isolated clump of native aspen near Halsey. Both the cuttings in the nursery and those set out directly in the sandhills callused well, and at the end of a few weeks 74 per cent had started leaves. Before roots started, the bark of all became blackened in definite patches which started at the callused surfaces or at buds, spreading till the entire cutting was involved. The planting was a total loss. Cuttings taken from the Pike Forest and planted in sand in a greenhouse at Washington, D. C., behaved in the same way, though part of them had been washed in mercuric chloride solution and the ends trimmed back with a sterile knife. In this case the cuttings which had started leaf growth blackened much earlier than those which had not. Two of these cuttings commenced to blacken at points remote from buds or visible wounds, an occurrence rarely observed. The cuttings planted at Washington were set in rows, and the living and dead mapped at intervals. No evidence was secured of the spread of the disease from foci. Inner bark cut from recently blackened areas both on diseased cuttings and in cankers found on living trees was planted in nutrient media. Beef agar, cornmeal agar, prune agar, aspen agar, and beef gelatin were employed. No organism appeared consistently and many of the plantings yielded no organism whatever. Rhizoctania hyphae were common on the surface of the diseased cuttings, but inoculation at wounds with artificial cultures of Rhizoctonia, Cytospora, and Macrosporium, the two latter taken from a canker on native aspen at Halsey, failed to give results. Pieces of recently blackened inner bark were fastened in slits in healthy bark without pathogenic results.

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[1] Determination by Mrs. H. E. Watkins.

The rapid blackening of bark is a common phenomenon in aspen killed from any cause, and therefore is not a diagnostic character for the work of any particular disease. It is entirely possible that no parasitic action was involved in this failure of the aspen cuttings. It does not seem likely that the Cytospora so commonly found was the cause of the death of the cuttings. Another possibility which seems to the writers worthy of consideration is that much of the bark killing, both on cuttings and on trunks and possibly twigs of older trees, may be due to a bacterial organism like the *Micrococcus populi* of Delacroix (3), which he was able to cultivate only in unheated poplar bark trituration. It seems especially likely that the cankers of type II may have been caused by an organism of this type.

## SUMMARY

Observations have shown quaking aspen in certain areas to be unusually subject to disease; trunk cankers of unknown origin seem to be especially important factors in shortening the life of the trees. *Fomes igniarius* is also an important factor in causing premature death of aspen in the Pike's Peak region.

Interesting but less important diseases are (1) a twig blight suggesting in appearance the fire blight of pear; (2) a leaf disease due to *Sclerotium bifrons*, E. & E., distributed from the Rocky Mountains to New England and also attacking Lombardy poplar but not *Populus grandidentata*; and (3) a rapidly spreading bark trouble which kills cuttings.

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# IRIS LEAF SPOT CAUSED BY DIDYMELLINA IRIDIS

W. B. TISDALE[1]

WITH SIX FIGURES IN THE TEXT

## INTRODUCTION

The most widespread disease of the German group of Iris in this country is the leaf spot caused by *Didymellina iridis* (Desm.) v. H. It is rivaled in seriousness only by the bacterial soft rot of the rhizomes. Because of the increasing prevalence of the disease in the vicinity of Madison, Wisconsin, and a lack of knowledge of the life history of the causal organism and of control measures, it has seemed a problem worthy of investigation. Accordingly, the writer undertook a study of the disease in October, 1916, at the University of Wisconsin. Since that time the life cycle of the fungus has been followed, the ascigerous stage found, and successful control measures worked out. In connection with this work various details relative to the morphology and the physiology of the organism were determined, which deserve mention although not a part of the main undertaking.

## THE DISEASE

### *History and geographical distribution*

The fungus causing Iris leaf spot has been known to mycologists in foreign countries for years, but it has attracted little attention from phytopathologists until more recently. Cooke (5) seems to have been first to consider the disease of any serious import. Later (6) the same author reported the disease as being very troublesome in several parts of England and Ritzema Bos (14) reported investigations upon a disease of Narcissus which he attributed to the Iris organism. The disease occurs in practically all of the European countries (6, 11), Canada (9), and the Cape of Good Hope, but no note was made of its importance in these regions.

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[1] The writer wishes to express to Dr. A. G. Johnson, at whose suggestion this investigation was undertaken, his sincere appreciation of the many valuable suggestions and helpful criticisms given during the progress of the work. He is further indebted to Dr. J. J. Davis and to Dr. C. L. Shear for their assistance in identifying the ascigerous stage of the organism, and to Dr. L. R. Jones for his assistance in the final preparation of the manuscript.

During the two years in which the problem has been under consideration, a partial survey has been made of the United States. Answers to questionnaires sent to plant pathologists of the agricultural experiment stations indicate that outside of Wisconsin the disease is prevalent only in California, Kansas, New York and Ohio. However, judging from the wide range of climatic conditions which obtain among these states, it is quite certain that the disease may develop in practically all regions where susceptible species of Iris are grown.

### *Hosts*

At Madison, Wisconsin, natural infection has appeared on only the broad-leaved or German Iris. The species of this group which have been found affected in this vicinity are[2] *Iris germanica*, *I. florentina* var. *albicans*, *I. variegata* var. *honorabilis*, and one unidentified species. Reports from European countries show that the same fungus crosses over to other species and genera of hosts and Cooke (5) reported it on Lychnis, a member of a widely separated family. Ritzema Bos (14) attributed a serious disease of several varieties of Narcissus to the same fungus. In 1888 Ellis collected, at Newfield, New Jersey, a fungus which he identified as *Heterosporium gracile* Sacc. on *Hemerocallis fulva*. The same organism has been reported by Lindau (11) as occurring on Freesia, Antholyza, Gladiolus, and Gemmingia. Since the only specimens readily available for examination were those of broad-leaved Iris, no comparative study of the organisms from the various hosts was made. Cross inoculation experiments to *Hemerocallis* sp., *Narcissus* Emperor, and Pheasant (Hort. varieties) with the organism from Iris were unsuccessful in every case. This would seem to indicate specialized races or even different species of the fungus on the various host genera.

### *Signs of the disease*

The disease has been observed on the leaves only, with infection most abundant on the apical half. It is first evident as minute brown spots surrounded by a watersoaked margin which later turns yellow (fig. 1). The spots occur on either surface of the leaf, but are not visible on the opposite surface until one or two days later. In the spring the spots enlarge slowly, requiring about thirty days to become ¼ inch in diameter. Later in the season, however, they enlarge much more rapidly and produce conidia after about two weeks. The size and the number of the spots vary with the species of host; they are about ¼ to ½ inch in diameter on *I. germanica* and smaller and more numerous on the other species. The older spots are oval or elliptical in shape, with the longer axis parallel to the veins. They may in some instances be so numerous as to kill the entire leaf early in the summer, but, as a rule, only a few of the leaves die before the blossoming season. It is not uncommon to find a striping of the leaves of *I. germanica*. This is due to dying of the tissue between the spot and the apex of the leaf. The older spots have a grayish center which is dotted with tufts of black conidiophores (fig. 2). Surrounding this center is a reddish brown border which is a deposit of some substance on the cell walls. The nature of this substance was not determined. The descriptions given by Cooke, Güssow, and Ramsbottom correspond very favorably with the above. It is, therefore, presumed that they were dealing with the same hosts.

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[2] Bailey's Standard Cyclopedia of Horticulture has been followed in the above classification and nomenclature.

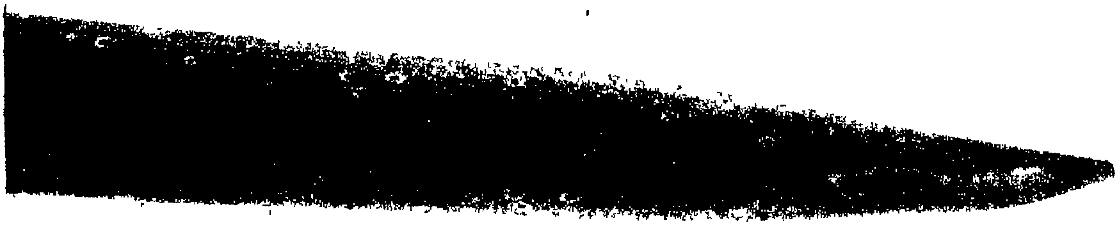


FIG. 1. IRIS LEAF SPOT TEN DAYS AFTER ARTIFICIAL INOCULATION

According to Ritzema Bos (14), the disease of Narcissus, for which he holds the same fungus responsible, attacks the plants in quite a different manner. He states that the first evidence of the disease is a yellowing of the margins of the leaf, which soon involves the whole leaf, thereby causing the sudden death of the affected parts. After the leaf dies its upper surface becomes covered with a blackish fungus growth. The leaves of Iris are very similar morphologically to those of Narcissus, and it seems unlikely that the same organism would produce only a spot on the former and completely destroy the leaves of the latter.

### *Economic importance*

*Didymellina iridis* reaches its most destructive stage at such a period in the growth of the plant that it is difficult to estimate the amount of damage incurred in any one year. Although the disease appears soon after the young leaves emerge in the spring, it does not become very noticeable until about the blossoming season, which in Wisconsin is the latter part of June. After this time the disease develops very rapidly and kills many of the leaves prematurely. This premature death of the foliage reduces the amount of food stored up in the rhizomes, and if the process continues from year to year the death of the plants will ultimately follow. The disease has not been followed closely enough and for a sufficient length of time to determine to what extent the vigor of the plants is thus reduced from year to year.

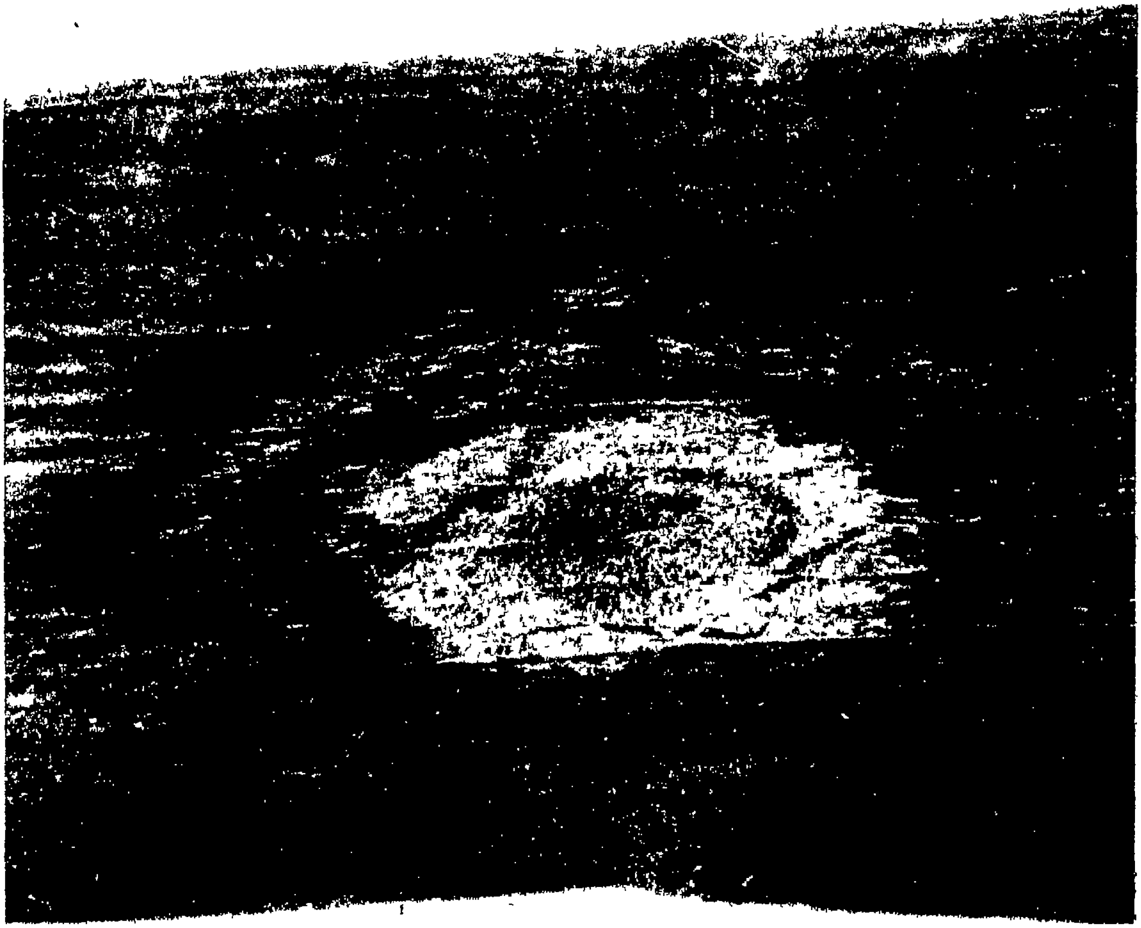


FIG. 2. OLD SPOT ON IRIS LEAF, SHOWING TUFTS OF CONIDIOPHORES
Natural infection. × 5

Cooke says (6): The leaf spot is one of the most troublesome and persistent diseases of Iris in England, the plants being killed in one or more years. Güssow (9), on the other hand, considers the disease of minor importance in Canada.

### *Pathological histology*

In all cases examined the germ tubes were found to enter the tissue through the stomata (fig. 3). Ramsbottom (13) states that he found penetration taking place both through the stomata and directly through the epidermis. Ritzema Bos (14) states that the fungus attacking Narcissus penetrates the tissue by both methods. Inasmuch as not a single case of direct penetration of the cuticle was found, it seems reasonable to believe that this mode of attack is the exception rather than the rule.

Upon entering the substomatal chamber the germ tube sends out branches which wedge between the cells, forcing them apart. Later the hyphae become intra-cellular, the cells collapse and the affected area dries out. The contents of the affected cells first turn yellowish and then gradually disappear. A reddish brown material accumulates on the cell walls in the margin of the affected zone, giving the characteristic brown margins. Small masses of hyphae develop in the substomatal chambers and give rise to tufts of conidiophores, which in crowding through the stomata tear the epidermis loose from the underlying tissue, thus giving the chracteristic gray color to the center of the spot. The hyphae seem to be restricted to the spot while the leaf is yet green, but after the leaf dies they spread throughout the tissue.

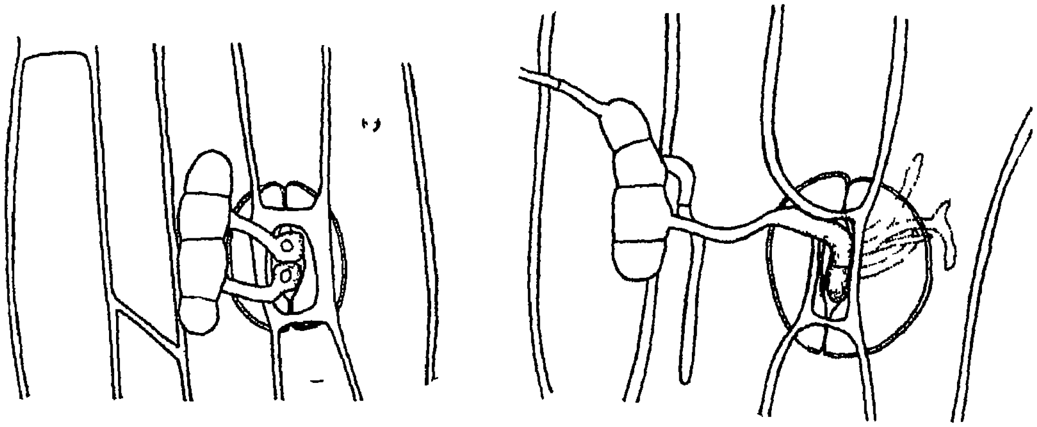


FIG. 3. DIFFERENT STAGES OF STOMATAL PENETRATION BY CONIDIAL STAGE OF DIDYMELLINA IRIDIS

Showing germination characteristics of conidia

## THE CAUSAL ORGANISM

### *Taxonomy*

*Conidial stage.* Early records of the fungus on Iris date back to 1881, when Saccardo (15) described the conidial stage of the organism and referred it to *Heterosporium echinulatum* (B. & Br.) Cooke. This specific name had previously been given by Berkeley (2) to the fungus attacking carnations. Later Saccardo (16) referred the fungus on Iris to *Heterosporium gracile* Wallr. He had, however, already referred the *Helminthosporium gracile* Wallr. to *Brachysporium* (16, p. 430). This, according to current description, is a very different fungus from the one under consideration here. On this account later authors as Cavara (3), Schroeter (17), Oudemans (12), Ramsbottom (13) and others have attributed the name *Heterosporium gracile* to Saccardo.

*Ascigerous stage.* During the course of these studies an ascomycete was found on the dead, infected leaves of *I. variegata* var. *honorabilis* and *I. florentina* var. *albicans*. Pure cultures obtained from single ascospores of this fungus have produced typical conidia, and the positive results of inoculation experiments with such conidia seem to be sufficient proof that the ascomycete under consideration is the ascigerous stage of *Heterosporium gracile.*

There seems to be no record in literature of any previous connection of *H. gracile* Sacc. with an ascigerous stage. Also, judging from description only, the ascigerous stage under consideration seems to differ from any species of sphaeriaceous forms attacking Iris.

Specimens were, therefore, sent to Dr. C. L. Shear at Washington, D. C., for identification. Upon comparison he found them to be identical with specimens distributed by Desmazieres in his Pl. Crypt. France, No. 94 (1853) on leaves and pods of *Iris pseudacorus* collected at Hermanville, France, by Roberge, and described as *Dothidia iridis* Desm. in Ann. Sci. Nat. Bot., Ser. 3, VIII Bd., p. 76, 1847. He also compared them with specimens collected by Roberge at Caen, France, and now in the herbariun of Michener.

Von Höhnel (10) in "Mycologische Fragmente," CCVII in Annales Mycologici, vol. XVI, nos. 1 and 2, 64–66, 1918, enters into a full discussion of the morphology and taxonomy of *Dothidia iridis* Desm. This author says (p. 66) that *Dothidia iridis* Desm. is a Didymella Sacc. (1878) without paraphyses and therefore belongs to a new genus which he calls Didymellina. He also claims that many species of Mycosphaerella belong to this genus. Accordingly, he applies the binomial *Didymellina iridis* (Desm.) v. H. and lists the following as synonyms:

Type species: *Dothidia iridis* Desm. 1847.
Synonyms: *Sacidium desmazierii* Montagne 1857
*Sphaerella iridis* Auersw. 1869
*Metasphaeria iridis* (Desm.) Sacc. 1883
*Mycosphaerella iridis* (Auersw.) Schroeter 1894
*Sphaerulina iridis* (Desm.) Berlese 1894
*Sphaerella desmazierii* (Mont.) Sacc. 1895
*Sphaerella desmazierii* (Roberge) v. H. 1910

Some confusion exists among mycologists as to the taxonomic position of some of the above forms, or else von Höhnel included forms which should not be considered as synonymous with the specimen originally described by Desmazieres. A comparison of specimens of *Sphaerella iridis* Auersw. and *Mycosphaerella iridis* (Auersw.) Schroet. in the herbarium of the Department of Botany of the University of Wisconsin with the fungus described in this article reveals a great difference in size of perithecia and of spores. The perithecia of these specimens measure only about 120 to 160 microns in length and the spores 4 to 5 by 12 to 17 microns. Two of the herbarium specimens which were on green leaves collected in June and September showed no evidences of a conidial stage. The spots had no definite reddish brown border and were more irregular in shape than those upon the material collected at Madison. As stated elsewhere, perithecia have been collected at Madison on only the dead leaves of Iris and only during the spring months.

## *Morphology*

The fungus, *Didymellina iridis* (Desm.) v. H., has two stages in its life cycle, namely, (1) the conidial stage parasitic on the leaves of Iris, and (2) an ascigerous stage which develops in the spring on the dead leaves infected during the previous season. The perithecia were found to be sterile two years out of the three during which the fungus was under observation and so appear to play a relatively unimportant part in the life cycle.

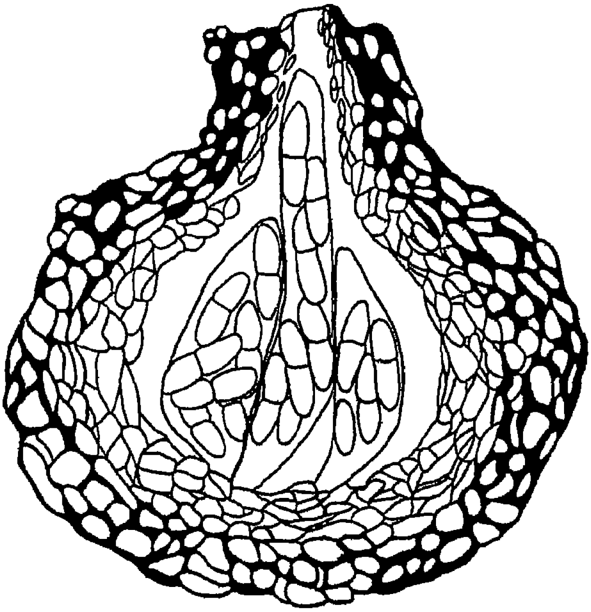
*Conidial stage.* Three or four weeks after primary infection occurs, tufts of conidiophores emerge through the stomata in the diseased spots and bear conidia. The conidiophores are olivaceous and more or less zigzag in shape at the upper end. The dimensions vary considerably with environmental conditions. The ones borne in the spots during the summer average about 11 by 70 microns, while those developing in the spring on the apices of sterile perithecia average about 12 by 150 microns.

The conidia are echinulate, concolorous with the conidiophores, cylindrical with rounded ends, two to three septate, slightly constricted at the cross walls, and range from 13.7 to 18.7 by 40 to 60 microns, averaging 16.8 by 50.8 microns. The range in size is quite variable in any one spot or in pure culture, and while the range is a little greater than that given by Saccardo and Lindau, it does not seem sufficient for specific separation.

*Ascigerous stage.* Perithecia scattered, smooth, globose, dark brown to black, 130 to 220 by 188 to 246 microns, averaging about 160 by 200 microns; subepidermal, with a papilliform ostiole which may bear a tuft of conidiophores under favorable weather conditions; walls pseudoparenchymatous, two to four cells thick (fig. 4).

Asci obclavate to oblong, hyaline, thin walled, rounded and obtuse above, tapering below into a short pedicel, or sessile, fasicled; 36 to 54 by 72 to 117 microns, averaging 49.9 by 97.2 microns; from 6 to 25 in a perithecium, without paraphyses (fig. 5).

Ascospores oval-elliptical, one septate, distinctly constricted, contents light greenish-yellow, wall slightly tinted; 10.5 to 16.2 by 30.6 to 54 microns, averaging 14.4 by 44 microns, slightly unequally two-celled; usually eight spores in an ascus.



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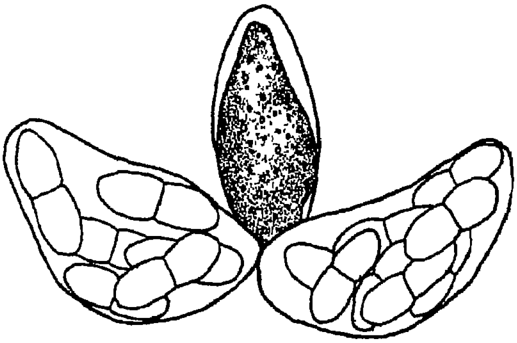

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Fig. 4. Didymellina iridis

Camera lucida drawing of a median section through a perithecium

Fig. 5. Asci of Didymellina iridis

Showing different stages of maturity in the same perithecium

## *Physiology*

*Cultural studies.* In pure culture the young hyphae are grayish-white, delicate and septate. On most media used this type of hyphae forms a dome-shaped, aërial colony within the first few days. After about five days the hyphae begin to form a thin, dark brown to black stroma on the surface of the medium from which olivacious conidiophores arise and produce conidia acrogenously. As each conidium approaches maturity it is pushed aside and the conidiophore continues to grow and produce more conidia until from two to four are produced. The conidia are very easily dislodged and are never found with more than two *in situ.*

The fungus grows well on most of the common laboratory media except beef-peptone agar. It also grows well on steamed stems of *Melilotus alba* and steamed leaves of Iris. As compared with the Fusaria and some other organisms, it does not grow very vigorously on any of the media, requiring about two weeks to cover the surface of a slanted tube of agar. It produced conidia more abundantly on potato agar, corn meal agar, and sterilized stems than on any of the other media used.

On steam sterilized stems of *Melilotus alba* and steamed Iris leaves the growth is at first similar to that on other media except that conidia are produced two or three days earlier. Perithecia begin to develop when the culture is about twelve days old, and reach their maximal size about ten days later. During the first week they contain abundant oil globules, but these finally disappear and the perithecia remain sterile. Such cultures were treated in many different ways in an attempt to stimulate the production of ascospores, but without success.

### *Germination studies*

*Conidia.* Conidia taken from a pure culture germinate readily on both solid and liquid media. Germ tubes are produced from any or all of the cells of each spore, and as many as three tubes may be produced from a single cell. The germ tubes from a single spore may develop simultaneously, or later in the middle than in the end cells. Spores germinate best and most rapidly at 20° to 26°C., as shown in table 1. At temperatures higher or lower than these germination takes place more slowly, and at 30° the tubes are densely vacuolate and grow very slowly.

TABLE 1

*The relation of temperature to the rate of conidia germination*

| TEMPERATURE | TIME REQUIRED FOR GERMINATION |
|---|---|
| *deg. C.* | *hours* |
| 4 | 21 |
| 8 | 6 |
| 13 | 4 |
| 17 | 3 |
| 22 | 2½ |
| 26 | 2½ |
| 30 | 11 |
| 35 | No germination |

The optimal temperatures (22–26°C.) commonly obtain out-of-doors during the latter part of May and June, when the disease is developing most rapidly.

*Ascospores.* Ascospores removed from perithecia and planted in water or on water agar germinate within about six hours. Development of the germ tube is in some cells immediately preceded by the appearance of a septum, and each of the three or four cells thus formed is capable of producing one or two germ tubes (fig. 6). Once started, the tubes grow fairly rapidly on potato agar and after five days a colony developing from a single ascospore produces conidia which are exactly similar to those of *H. gracile.* The colonies developing from single ascospores are likewise similar to those developing from conidia, when grown on the same kind of medium.

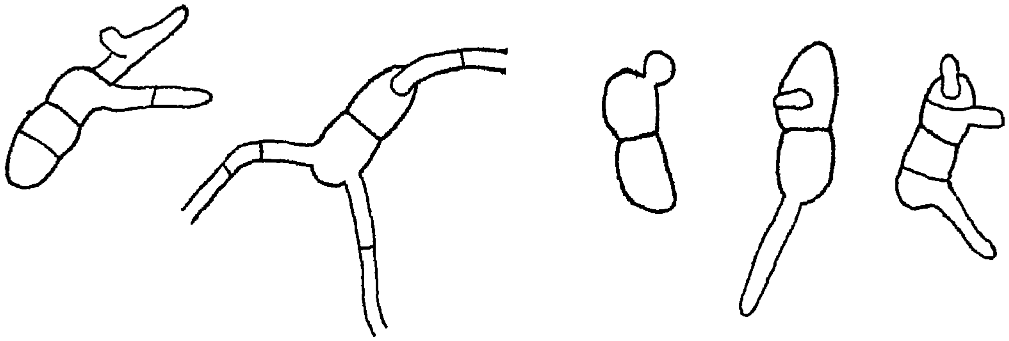

FIG. 6 GERMINATING ASCOSPORES OF DIDYMELLINA IRIDIS

### *Overwintering and life history of the organism*

The causal organism of Iris leaf spot passes the winter in the mycelial stage in the old infected leaves, and produces conidia and perithecia early the following spring. During the course of these studies no viable conidia were found in the field from December to March. However, an examination of the dead leaf tissue revealed the presence of dark brown, thick walled hyphae and when the leaves were brought into the laboratory and kept in a moist chamber at room temperature for two or three days abundant conidiophores and conidia developed in the old spots. Conidia developed in culture at 8°C. in fifteen days, and were found outside in March, following a few days of warm weather. This seems to indicate that successive crops of conidia are produced throughout the winter in sections having a milder climate than that of Wisconsin. Since the conidia germinate within twenty-four hours at 4°C., it seems reasonable to suppose that, unless subjected to extremely low temperatures, they are short lived in nature.

Late in February and early in March small masses of dark colored hyphae appear beneath the epidermis in lesions on old leaves and later develop into perithecia. The outcome of the perithecia seems to be dependent upon weather conditions. In the spring of 1917 they matured about April 1, but in all cases were found to be sterile. Most of these sterile perithecia bore a tuft of four to twelve conidiophores on the apex, or ostiole. This peculiar manner of fructification is probably due to the fact that the sudden change from cold to warm temperature was unfavorable for the production of asci. During the spring of 1918, which was somewhat similar to that of 1917, a few perithecia were found to contain asci and ascospores. The spring season of 1919 opened rather early and was followed by intermittent cool periods which continued well into April. Under such weather conditions one would expect the fungus to react quite differently than it did the two previous years with different weather conditions. At any rate, the old leaves which were taken from the field about March 30 bore numerous perithecia containing mature asci and ascospores. These fertile perithecia bore no conidiophores at the apex, as did the sterile ones the two previous years. Such erratic occurrence of ascospores indicates that they play a minor part in the early infection of new leaves. On account of the difficulty experienced in obtaining a number of ascospores free from segments of hyphae and conidia, no inoculations were made with them directly.

Since there is always an abundant supply of newly developed conidia by the time the young leaves appear, it is probable that they serve as the chief source of primary infection. In infected beds the new leaves come into direct contact with the old infected leaves and thus, incidentally, are sprinkled with a plentiful supply of viable conidia. Perhaps rain, too, aids in distributing the spores. The primary spots begin to appear about the middle of April and after about a month, depending upon the weather conditions, are producing conidia, which, in turn, serve to continue the infection. After the blossoming season the spots become numerous on all susceptible species and many of the leaves are thus killed down before frost.

## INOCULATION EXPERIMENTS

The inoculation experiments with the Iris leaf spot organism were conducted during the spring seasons of 1917 and 1918. Practically all of the experimental work was done in the greenhouse to insure against outside infection. Rhizomes from which all of the dead infected leaves were removed were brought in from the gardens during the winter and planted in pots in the greenhouse. These plants grew vigorously and apparently normally, and no signs of disease developed on the uninoculated plants at any time. The experiments were conducted along two lines, namely: (1) inoculation of the varieties of Iris which were attacked by the fungus outside, and (2) cross inoculation to *I. siberica, Narcissus* varieties Emperor and Pheasant and one unidentified species of Hemerocallis. After inoculation the plants were kept in a moist chamber for about thirty-five hours and then placed in a greenhouse room where the temperature was held fairly constant at about 22°C.

Three methods of inoculation were used, namely: (1) insertion of fragments of mycelium into the leaf tissue with a sterile needle, (2) sowing the conidia in drops of sterile water on marked areas of the leaf surface, and (3) spraying the plants with a sterile water suspension of conidia.

TABLE 2

*Results of the inoculation experiments with H. gracile*

| KIND OF PLANT | KIND OF INOCULUM | NUMBER OF PLANTS INOCULATED | NUMBER OF PLANTS INFECTED | PERIOD OF INCUBATION | INFECTION RESULTS |
|---|---|---|---|---|---|
| | | | | *days* | |
| I. germanica | Mycelium | 3 | 3 | 2 | + |
| I. germanica | Mycelium | 4 | 4 | 2 | + |
| I. variegata var. honorabilis. | Mycelium | 3 | 3 | 2 | + |
| I. variegata var. honorabilis. | Conidia | 2 | 2 | 5 | + |
| Hemerocallis sp. | Conidia | 2 | 0 | | − |
| Hemerocallis sp. | Mycelium | 2 | 0 | | − |
| I. siberica | Conidia | 4 | 0 | | − |
| I. siberica | Mycelium | 3 | 0 | | − |
| Narcissus var. emperor | Mycelium | 2 | 0 | | − |
| Narcissus var. emperor | Conidia | 4 | 0 | | − |
| Narcissus var. pheasant. | Conidia | 3 | 0 | | − |
| Narcissus var. pheasant | Mycelium | 2 | 0 | | − |
| Hemerocallis sp. | Mycelium | 3 | 0 | | − |
| I. germanica | Conidia | 3 | 3 | 8 | + |
| I. germanica | Conidia | 6 | 6 | 5 | + |
| I. florentina var. albicans. | Conidia | 2 | 2 | 5 | + |
| I. florentina var. albicans. | Conidia (ascospore strain) | 3 | 3 | 6 | + |
| I. germanica | Conidia (ascospore strain) | 2 | 2 | 8 | + |

In practically every case all three methods gave positive results when susceptible species of Iris were used. Previous to inoculation with conidia, the heavy bloom was removed from some of the leaves by lightly rubbing them with a piece of clean cheesecloth. Water adhered better to the leaves treated in this manner than to untreated leaves and they always showed heavier infection. The results of the inoculation experiments are given in table 2.

The inoculation experiments show that the varieties of German Iris are very easily infected, while *I. siberica*, Narcissus, and Hemerocallis are evidently immune. Where mycelium was the source of inoculum, infection took place very rapidly with susceptible hosts and after about six days the spots were about ½ inch in diameter and were bearing conidia. Infection from conidia occurred in from five to eight days and developed very slowly at first, attaining a diameter of from 1 to 3 mm. in thirty days. The lesions developed more rapidly on plants which were placed outside of the greenhouse as soon as infection took place.

The failure of attempts to produce infection in Narcissus and Hemerocallis suggests the probability of different strains or species of the fungus. The varieties of Narcissus used in these experiments are listed by Ritzema Bos (14) as being susceptible to the disease with which he worked and which he attributed to *H. gracile.* This organism has also been reported by mycologists as occurring upon Hemerocallis both in America and in Europe. No strains of the organism were obtained from any other host than the species and varieties of German Iris herein mentioned and so far as these experiments justify conclusions, it is very likely that cross inoculation experiments with the organism from different hosts will show the existence of different strains or even of different species.

## CONTROL MEASURES

The writer has been unable to find any record in literature of successful control measures for the Iris leaf spot. Güssow (9) and Cooke (6) each recommended the removal of the dead infected leaves but did not show any evidence that the method had been successfully employed. Ramsbottom (13) stated that the limeloving species of Iris were the only ones affected with the leaf spot and that they were attacked only when grown on soil deficient in this substance. He therefore planned some experiments based on this theory, but for some reason never reported upon execution of them.

After the development of the fungus had been carefully followed during the winter and spring of 1917 at Madison, Wisconsin, it was thought advisable to try two methods of control, namely: (1) removal of dead leaves prior to the development of young leaves in the spring, and (2) protection of plants with a coating of some copper fungicide during their early development.

On March 30, 1917, the dead leaves were removed from two small beds of *I. germanica* and one of *I. variegata* var. *honorabilis*. However, by this time the leaves of *I. germanica* were from 1 to 2 inches long and conidia were being produced in small numbers on the dead infected leaves. One of the beds of *I. germanica* was sprayed with 4-4-50 Bordeaux mixture. At the same time and subsequently at intervals of two weeks until June 20, the spray mixture was applied to one small bed of each of the above named species from which the leaves had not been removed. No signs of infection developed on the plants in any of the three beds from which the leaves were removed until late in the summer, and then only a few lesions appeared. It is likely that the spores causing this infection came from nearby diseased beds. The disease developed somewhat later on the sprayed beds from which the leaves were not removed than upon the unsprayed controls, but was more abundant and widespread than upon the beds from which the leaves had been removed. This indicates that primary infection was prevented but that the plants were not continuously protected by the spray mixture. On account of the heavy bloom on the Iris leaves, it is a difficult problem to get the spray mixture to adhere uniformly. Also, as the plants approach the flowering stage it becomes very difficult to reach all parts of the leaves with the spray solution. Since the conidia are present on the dead leaves until summer, the plants would have to be kept continuously covered with the spray in order to insure protection.

Since the above difficulties had been encountered in 1917, it was decided to resort only to the removal of dead leaves as a possible means of control in 1918. Immediately after the snow had melted, two beds of *I. germanica*, the dimensions of which were about 8 by 10 feet, received this treatment. These plots were located in the horticultural gardens and far enough away from other plots to insure against infection from other sources. Previous to June 15 no signs of the disease had appeared in either of the treated plots, while in the check plots abundant infection was found by the first of May. Furthermore, Professor L. R. Jones employed in his private garden, which was planted with *I. germanica* and *I. variegata* var. *honorabilis*, the method used in these experiments, with the result that no signs of the disease had appeared in either of these beds up to June 15. Probably this method will prove effective for controlling the disease in all regions where the leaves die down during the winter. Under these climatic conditions the dead leaves are easily removed in a practical way by simply giving the plants a thorough raking or "combing" with a steel garden rake. In the warmer climates where the leaves remain green over winter, the method may have to be modified or used in combination with some spraying program.

## SUMMARY

1. Leaf spot is one of the most widespread diseases of the broad-leaved or German group of Iris in the vicinity of Madison, Wisconsin.

2. The disease is present wherever the susceptible species of Iris are grown. In the United States it has been reported as of considerable importance in California, Kansas, New York, Ohio, and Wisconsin. Cooke reports it as one of the most serious and troublesome diseases of Iris in England.

3. The groups of Iris other than the broad-leaved group and also the species of Narcissus and Hemerocallis used in the experiments are evidently immune from the disease.

4. The disease is caused by a fungus the conidial stage of which has been known for some time as *Heterosporium gracile* Sacc. The ascigerous stage of the organism has been found at Madison, Wisconsin, and has been referred to *Didymellina iridis* v. H.

5. The fungus overwinters in the mycelial stage in the dead leaves. Perithecia develop early in the spring, but do not always produce asci, apparently on account of the weather conditions. Sterile perithecia bear conidia on the apex.

6. An abundant crop of conidia, always present early in the spring, serves as the chief source of primary infection.

7. The only mode of penetration found during the experimental work at Madison was through the stomata.

8. The removal of dead infected leaves before the young leaves appear in the spring promises to be a successful means of control.

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# THREE WINTER WHEAT VARIETIES RESISTANT TO LEAF RUST IN KANSAS[1]

LEO E. MELCHERS AND JOHN H. PARKER

WITH THREE FIGURES IN THE TEXT

## INTRODUCTION

Observations on the occurrence of the leaf rust of wheat, *Puccinia triticina* Eriks, have been made at Manhattan and in other parts of Kansas with particular reference to the comparative susceptibility of varieties during the seasons of 1914–1919, inclusive. These observations have shown that in certain localities leaf rust may be present in quantities sufficient to cause actual damage (3).

The leaf rust of wheat as it occurs in Kansas may or may not show the same biologic specialization as the leaf rust occurring in other regions. There is also, the possibility that more than one biologic form has been present in the experimental plots during the six-year period in which the data were obtained, though the results with the varieties do not definitely indicate it. Some caution, at least, is necessary in presenting and interpreting the effects produced by any cereal rust in any one locality, in view of the known existence of several distinct biologic forms of stem rust of wheat, as described by Stakman and Piemeisel (6), Melchers and Parker (5), and Levine and Stakman (2).

## WINTER WHEAT VARIETIES RESISTANT TO LEAF RUST

In the investigations conducted at Manhattan, resistance to leaf rust has been observed in the same varieties which show a marked degree of resistance to one form of stem rust, namely, *Puccinia graminis tritici* (4). These varieties are Kanred, P762 (C. I. 5146) (Kansas No. 2401); P1066 (C. I. 5879) (Kansas No. 2415); and P1068 (C. I. 5880) (Kansas No. 2414). All these varieties belong to the Crimean hard winter wheat group and are bearded with glabrous, white, smooth chaff and hard, red kernels.

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[1] Coöperative investigations between the Kansas Agricultural Experiment Station and the Office of Cereal Investigations, Bureau of Plant Industry, United States Department of Agriculture. Published with the approval of the Director, as a contribution of the Department of Botany and Plant Pathology, Kansas Agricultural Experiment Station, paper No. 30.

Kanred (1) has been distributed by the experiment station to farmers and is probably the best variety of hard winter wheat now available for Kansas conditions. The other two varieties seem fully as promising in every

TABLE 1

*Data showing infection by leaf rust on three varieties of hard, red winter wheat and on adjacent rows of other winter wheats, at Manhattan, Kansas, during the six years from 1914 to 1919, inclusive*

| YEAR | VARIETY | AVERAGE PERCENTAGE | | MAXIMUM ON ANY VARIETY |
|---|---|---|---|---|
| | | On resistant varieties | On varieties in adjacent rows | |
| 1914 | P762 Kanred | 10.0 | 40 and 65 | 90 |
| 1914 | P1066 | 7.0 | 70 and 50 | |
| 1914 | P1068 | 7.0 | 40 and 60 | |
| 1915 | P762 Kanred | 10.0 | 40 and 25 | 65 |
| 1915 | P1066 | 5.0 | 25 and 40 | |
| 1915 | P1068 | 5.0 | 40 and 25 | |
| 1916 | P762 Kanred | 15.0 | 25 and 30 | 70 |
| 1916 | P1066 | 5.0 | 30 and 30 | |
| 1916 | P1068 | 5.0 | 30 and 30 | |
| 1917 | P762 Kanred | 10.0 | 40 | 65 |
| 1917 | P1066 | 5.0 | 37 and 32 | |
| 1917 | P1068 | 3.0 | 32 and 25 | |
| 1918* | P762 Kanred | 5.5 | 77 and 79 | 95 |
| 1918* | P1066 | 8.5 | 82 and 76 | |
| 1918* | P1068 | 9.0 | 70 and 78 | |
| 1919† | P762 Kanred | 6.7 | 79 and 73 | 90<br>With very heavy infection on the sheath and peduncles |
| 1919† | P1066 | 6.6 | 66 and 66 | |
| 1919† | P1068 | 5.0 | 71 and 66 | |

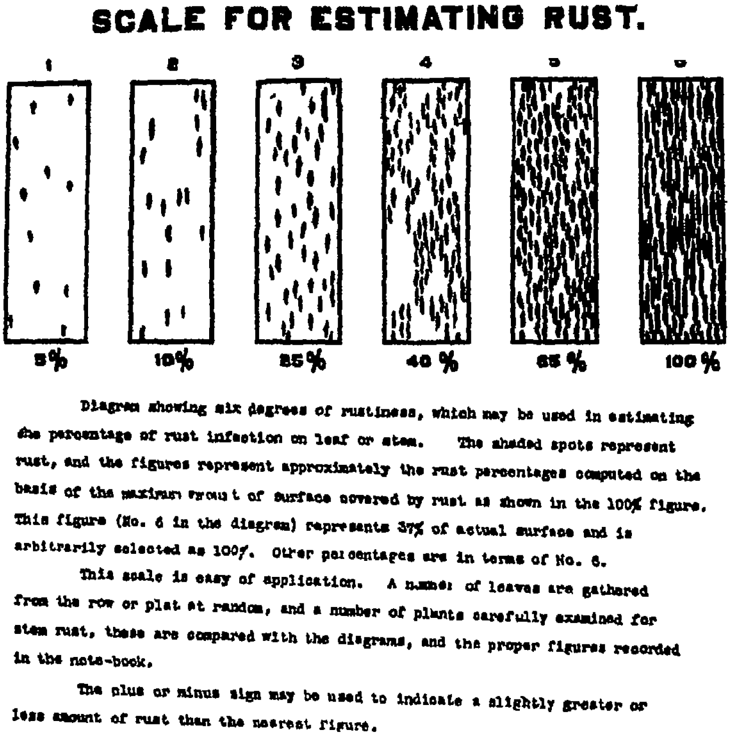
| | *per cent* |
|---|---|
| Six-year average for P762 (Kanred) | 9.5 |
| Six-year average for P1066 | 6.2 |
| Six-year average for P1068 | 5.7 |

* The rust percentages for this year are averages of a larger number of rows (pure line selections) grown in the rust nursery.

† The rust percentages for this year were taken from three series of variety plots at the agronomy farm, in addition to those obtained in the botany rust nursery. All varieties, with the exception of these three, showed from a trace to very heavy infection on the sheaths and peduncles.

respect, judging from records on yield, winter survival, and other characters obtained in the agronomy nursery. They have not, however, been compared in field plots, or in the cooperative experiments with farmers in the different sections of the state.

The observations on leaf rust infection presented in this paper were made in the rust nursery maintained by the Department of Botany and Plant Pathology, in the agronomy nursery and in commercial fields. The figures in table 1 are averages from a large number of plants, taken from rows in different parts of the nursery. The estimates of the amount of infection were made in accordance with the scale for estimating rust which is used by the Office of Cereal Investigations of the United States Department of Agriculture and is shown in figure 1.



SCALE FOR ESTIMATING RUST.

Diagram showing six degrees of rustiness, which may be used in estimating the percentage of rust infection on leaf or stem. The shaded spots represent rust, and the figures represent approximately the rust percentages computed on the basis of the maximum amount of surface covered by rust as shown in the 100% figure. This figure (No. 6 in the diagram) represents 37% of actual surface and is arbitrarily selected as 100%. Other percentages are in terms of No. 6.

This scale is easy of application. A number of leaves are gathered from the row or plat at random, and a number of plants carefully examined for stem rust, these are compared with the diagrams, and the proper figures recorded in the note-book.

The plus or minus sign may be used to indicate a slightly greater or less amount of rust than the nearest figure.

FIG. 1. SCALE USED FOR ESTIMATING RUST PERCENTAGES

It will be seen that the amount of leaf rust on the three varieties described as resistant was consistently less than on other varieties grown in adjacent rows and very much less than the maximum amount recorded on the most susceptible varieties grown in the nursery. It should be added that, during each season, the maximum percentage of leaf rust recorded was not confined to one or two varieties, but that there generally were a number on which the amount of infection reached this maximum, while a still larger number bore leaf rust percentages closely approaching the maximum figure.

The six-year average infection on Kanred, P762, is 9.5 per cent; on P1066, it is 6.2 per cent; and on P1068, only 5.7 per cent. These averages are remarkably low and indicate that the three varieties possess a high degree of resistance, even when grown under optimum conditions for infection. These conditions may be considered as the final test of the resistance or susceptibility of any variety, at least as far as its immediate practical value is concerned.

Figure 2, *A* and *A′*, shows the characteristic infection of leaf rust on the leaves of a susceptible variety, P706 (Kansas No. 2382), Improved Turkey. The uredinia are numerous and entirely normal. The leaves from which the illustrations were made were typical of this variety and were collected from plants in rows adjacent to P762, P1066, and P1068. Figure 2, *B* and *B′*, shows leaves of the resistant variety Kanred, P762. There are few uredinia, but many light yellow or green flecks, indicating the presence of rust hyphae, but failure to form uredinia. Figure 3, *C* and *C′*, shows leaves of another resistant variety, namely, P1066. There are few uredinia but many sharp flecks. Figure 3, *D*, and *D′*, shows two leaves from the third resistant variety, P1068. There are few uredinia, but the flecks, characteristic of the resistant varieties, are very distinct. The discolored brown areas of leaf tissue are due to the drying of the leaf from other causes and should not be confused with the leaf rust. The specimens shown in the illustrations were collected in June, 1918, from plants grown in the rust nursery, where a uniformly heavy infection of leaf rust was present. The varieties were approaching maturity and the leaf rust infection had unquestionably attained its maximum development.

In 1916 leaf rust was very prevalent in many wheat fields in the state and the infection was particularly conspicuous because it occurred on the peduncles or necks and sheaths of the plants, as well as on the leaf blades. A field of P706, Improved Turkey, at the agronomy farm near the college, showed an actual reduction in yield which, in the opinion of members of the Department of Agronomy, resulted from the heavy attack of leaf rust. A field of P762, Kanred, grown on the same farm had only a light infection on the leaf blades, recorded as 15 per cent, and no uredinia on the necks. The yield of this variety was not appreciably affected. The resistance to both leaf and stem rust was one factor which aided the Department of Agronomy in deciding to distribute P762, Kanred, instead of P706, Improved Turkey, known to be susceptible to both rusts.

Additional evidence on the resistance of P762, Kanred, was obtained in the agronomy nursery during the summer of 1918. An experiment on the date of planting as related to the habit of growth was located near the other nursery plantings and contained the following varieties: Defiance Winter (Kansas No. 373), Currell (Kansas No. 2406), Turkey (Kansas

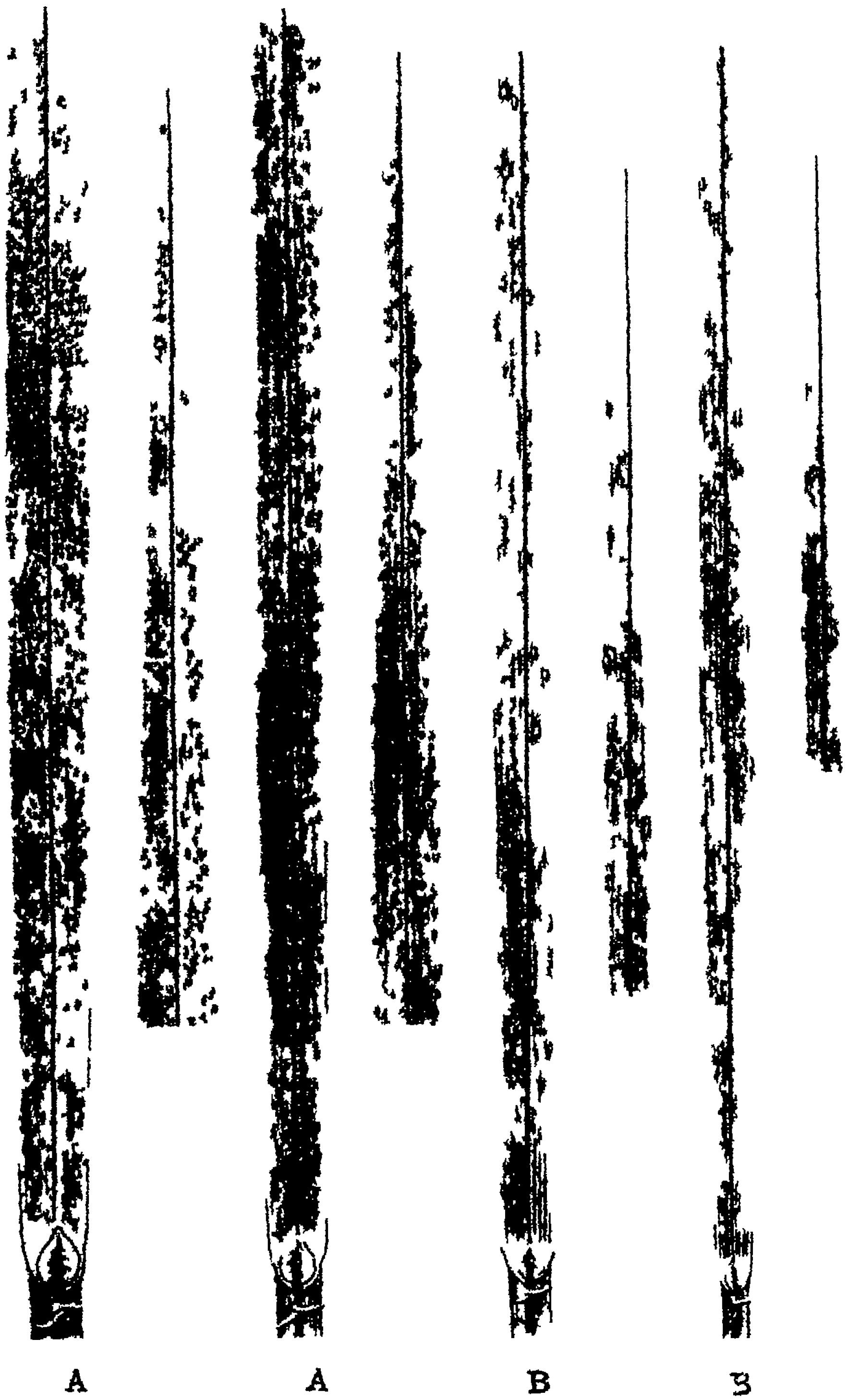


FIG. 2. A. The characteristic infection of leaf rust on the leaves of a susceptible variety P 06 (Kansas No. 2382) Improved Turkey. B. the leaves of the resistant variety P 62 (Kansas No. 2401) Kanred. Note only a few uredinia but numerous flecks denoting resistance.

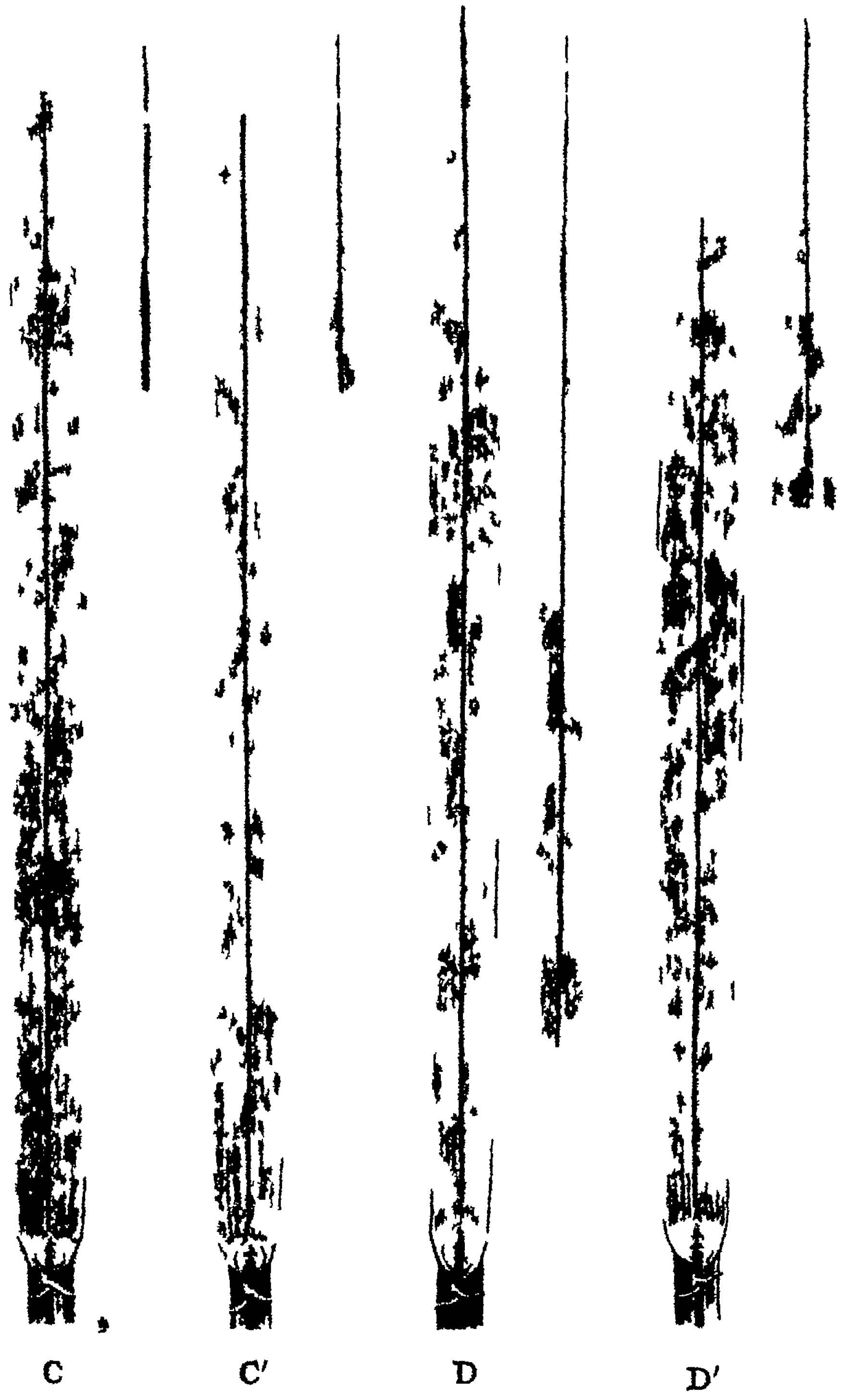


Fig. 3. C, leaves of the resistant variety P1066 (Kansas No. 2415); note few uredinia but many sharp flecks. D, leaves of a third resistant variety P1068 (Kansas No. 2414). Note few uredinia but many flecks which are characteristic of the resistant varieties. The discolored brown areas of the leaves are not due to leaf rust infection.

No. 570), Kanred (Kansas No. 2401), Fultz (Kansas No. 2156), and Nebraska No. 28 (Kansas No. 34). These were planted on the first of each month and in the order mentioned. The rows of Kanred were located between Turkey (Kansas No. 570), a hard wheat, and Fultz (Kansas No. 2156), a soft wheat. Both of the latter varieties are known to be susceptible to leaf rust. The plantings which were made on June 1 and July 1 were making vegetative growth during the ripening of the wheats in the regular nursery and after they were harvested. There was an abundance of natural infection of leaf rust in the nearby regular nursery plantings, and these were scattered and continued to spread on the green plants in the date of planting experimental plots. A heavy infection was present in these rows throughout the late summer and early fall and the amount of leaf rust on the plants of Kanred was not more than 20 per cent, while that on the adjacent rows and all the other varieties, varied from 50 to 85 per cent. There were some uredinia on the leaves of the Kanred plants, but flecks were much more numerous. These observations made on late plantings materially strengthened the evidence of the resistance of P762, Kanred, for a variety sometimes shows resistance when planted at the normal time, but fails to maintain this resistance when planted on a later date.

In addition to the observations made at Manhattan, there has been evidence from the reports of cooperating farmers that fields of Kanred grown in various parts of the state are much less affected by leaf rust than are fields of other varieties commonly grown on the same farm. These reports were definitely confirmed the past season. In the Kanred seed inspection work which was carried out in fields of Kanred wheat which were grown in Kansas the past season, a thorough examination was made for both leaf and stem rust. A most severe epidemic of leaf rust occurred in all parts of the state during the spring of 1919, which afforded the best opportunity to see what the behavior of Kanred would be in Kansas when grown on a commercial scale. Specimens were gathered from about 46 counties representing more than 500 fields. Notes were taken on the percentages of leaf rust, not only of Kanred, but of all adjoining fields of wheat of other varieties. The percentages for Kanred were strikingly uniform, ranging between 8 and 10 per cent, while the percentages of leaf rust for adjoining fields of other varieties which served as checks, varied from 20 to 95 per cent, the majority ranging close to the latter figure. The sheaths of Kanred plants were always entirely free of any leaf rust sori, while all other varieties showed from slight to heavy infection. It has not been possible to obtain as extensive records of P1066 and P1068, since these varieties have not been distributed to farmers but the indications are that they will be just as resistant as Kanred.

Experimental plantings of the three varieties made at certain stations in other states have also had a lighter infection of leaf rust than local varieties grown under the same conditions. These varieties must be grown at these and other stations for a series of years, however, before final conclusions as to their resistance in these localities can be reached.

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# BRIEFER ARTICLES

## THE DISTRIBUTION OF BUCKEYE ROT OF TOMATOES

J. L. WEIMER

The buckeye rot of tomato fruit, caused by *Phytophthora terrestria* was first described by Sherbakoff from Florida in 1917. He had knowledge of its existence in southern Florida since 1915, and preserved specimens found in the laboratory of the Florida Agricultural Experiment Station indicated that it had been prevalent as early as 1911. This disease was reported to the Plant Disease Survey from Arkansas and Porto Rico in 1917 and from Texas and Louisiana in 1918. Bruner states that what he supposed to be *Phytophthora terrestria* was found causing a rot of green tomatoes in Cuba in 1918. So far as available records show, this disease is present only in tropical or subtropical regions. The purpose of this note is to record its occurrence in Alexandria County, Va., on the United States Department of Agriculture experimental farm at Arlington, Virginia, in 1919.

During the latter part of August several green tomatoes showing the typical brown lesions with the concentric rings, as illustrated by Sherbakoff, were found. Other specimens were obtained at different times throughout the remainder of the season not only in this but also in two other patches, each being a considerable distance from the other. In the patch where the first specimens were found considerable damage resulted to the fruit in contact with or very close to the soil. In no case, however, was the fungus found causing a disease of the stems or leaves. Isolations were made and a Phytophthora conforming to the description of *P. terrestria* obtained, and its parasitism proved by inoculations made from pure cultures into tomatoes. This organism was also compared with an authentic culture of *P. terrestria* from Florida.

Buckeye rot of tomatoes, then, is not restricted to the extreme Southern States, as previous reports on its occurrence might seem to indicate.

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# VARIETAL SUSCEPTIBILITY TO FALSE BLOSSOM IN CRANBERRIES

## S. B. FRACKER

On a recent examination of nursery inspection reports, the writer was impressed with the marked difference between the varieties of cranberries in the amount of "false blossom" found on the bog. The variation was so striking that an analysis of the reports for the past five years was made and it is presented herewith.

The nursery inspection office of the State Department of Agriculture of Wisconsin was requested in 1915 to provide for the inspection of cranberry vines and to grant certificates in the same way that other plants and plant products for propagation are handled. An accusation that certain growers were planning to dispose of vines in which false blossom was present was the original cause of this action, and this disease condition has been the principal cause for refusing to grant inspection certificates to cranberry growers, although occasionally the presence of insect pests has resulted in similar action.

The so-called "false blossom" condition has been described by Shear[1] and by Jones and Shear.[2] In Wisconsin it is the cause of a serious loss in production. The symptoms are the development of what appears to be a blossom, followed by a green shoot instead of a berry. The pedicel of the flower remains erect instead of bending over, as normal blossoms do, and it is said to be distinguished from lack of pollination in this manner.

No organism responsible for the trouble has been isolated, and there is little evidence to show that the condition is an infectious disease. It is fairly common in Wisconsin bogs which have been planted to berries of the so-called "Berlin" group, although varieties not belonging to this group have also developed the same condition. An idea of its prevalence may be gained from the fact that it has been necessary to refuse permits for the sale of plants from an average of 54 acres per year or about 20 per cent of the 252 acres inspected.

The acreage of the varieties inspected and the per cent of false blossom found are shown in the following table.

Two extremes of susceptibility are apparent. The native Wisconsin varieties, Bell and Cherry and Metallic Bell are so uniformly victims of this condition that the source of plants of these varieties has become greatly reduced. In fact the situation is shown too conservatively in the table, for large acreages in which false blossom was found in 1915 have been withdrawn from inspection and no plants have since been sold from these bogs. Less than half an acre of Metallic Bell a year, 6 per cent of the inspected area of this variety, has been licensed for sale. The 44 per cent of the Bell and Cherry acreage shown represents less than 10 per cent of the bogs from which Bell and Cherry might be sold were it not for false blossom.

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[1] Shear, C. L. Cranberry diseases. Bul. 110, Bur. Plant Ind., U. S. D. A. 1907.

[2] Jones, L. R., and Shear, C. L. A report upon false blossom and other cranberry maladies. Rept. 27th Anl. Mtg. Wis. Cranb. Gr. Assn. 1914, pp. 13, 14.

At the other extreme are the more vigorous growers, McFarlin, Searls Jumbo, and Prolific, with practically clear records. Bennett Jumbo seems to be comparatively resistant while the acreages of Potter's Favorite, Early Ohio, and other varieties examined have been too small to show positive results.

This varietal susceptibility might be considered as due to the different cultural conditions under which the different varieties are grown, but this appears unlikely owing to the large number of different bogs upon which each variety was observed. It is the opinion of Mr. O. G. Malde, who has made all the cranberry inspections for the department, that improper cultural conditions from which Bell and Cherry and Metallic Bell suffered years ago have resulted in deteriorated stock, but whether false blossom could be developed and inherited under such circumstances is questionable. Cultural conditions do greatly modify the severity of the attack, for the disease is much more common on wet bogs than on the drier type. The application of commercial fertilizer and sanding of the bogs have also resulted in an improvement even where false blossom was already present.

*Table showing false blossom acreage*

| VARIETIES LISTED IN ORDER OF TOTAL ACREAGE INSPECTED | AVERAGE ACREAGE INSPECTED ANNUALLY | AVERAGE ACREAGE INFECTED ANNUALLY | PER CENT DISEASED | TOTAL NUMBER OF INSPECTIONS MADE 1915-1919 | NUMBER OF OWNERS OF INFECTED PLANTS |
|---|---|---|---|---|---|
| Searls Jumbo | 79 | 0 | 0 | 18 | 0 |
| Bell and Cherry | 60 | 34.0 | 56 | 17 | 8 |
| Bennett Jumbo | 50 | 11.0 | 22 | 16 | 8 |
| McFarlin | 37 | 0.2 | 1 | 14 | 1 |
| Prolific | 7 | 0.4 | 6 | 16 | 1 |
| Early Ohio | 7 | 1.4 | 20 | 4 | 2 |
| Metallic Bell | 6 | 5.6 | 93 | 14 | 12 |
| Others and mixed | 6 | 1.6 | 27 | 16 | 5 |
| Total | 252 | 54.2 | 21 | | |

Even though the cause may be a physiological one, plants from an infected bed continue to develop false blossom after being transferred to new locations. The loss usually then becomes greater from year to year and total crop failure has sometimes resulted, followed by the abandonment of the bogs. Under these circumstances the nursery inspection office must adopt the same policy in providing for cases of false blossom as if the disease were proven to be of an infectious nature.

We are recommending to cranberry growers: (1) that they choose resistant varieties of strong, vigorous growth for new plantings; (2) that they take vines for new plantings from beds without a trace of false blossom; (3) that they adopt cultural methods which will secure the largest returns in healthy bogs, for these are also the best ones for assisting affected plants to overcome the false blossom condition.

STATE DEPARTMENT OF AGRICULTURE
MADISON, WISCONSIN

# OBSERVATIONS ON SEED TRANSMISSION OF THE CABBAGE BLACK ROT ORGANISM

J. C. WALKER AND W. B. TISDALE

Since its presence on cabbage seed after harvest was first demonstrated by Harding, Stewart, and Prucha,[1] the transmission of the cabbage black rot organism (*Bacterium campestre* (Pam.) Smith) in this manner has been generally assumed. These investigators further showed that when seeds were bathed in a water suspension of the organisms and allowed to dry, some of the latter were still viable and pathogenic after eleven months. Since conclusive evidence of the organism living over from one season to the next on naturally infected seed is either lacking or very meager in the literature, observations strengthening this point and also giving evidence of the efficacy of seed disinfection are offered in this note.

The disease appeared in epiphytotic form in a large number of fields in the Racine, Wisconsin, district in 1919. When first observed on August 14 in a field of Copenhagen Market variety, it was well advanced and of such long standing that many plants had been prevented from heading. Part of this same field was planted with a late variety which came from a different source and these plants were practically free from disease. Further survey of this region during the remainder of the season showed many fields with general infection, while in numerous instances another variety in the same field was entirely healthy.

Inquiry as to the source of seed in each case revealed the fact that in every instance where black rot developed in epiphytotic form the seed had been secured from the same local seedsman. This dealer, it was learned, had distributed two lots of 1918-grown seed in the spring of

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[1] Harding, H. A., Stewart, M. C., and Prucha, M. J. Vitality of the cabbage black rot germ on cabbage seed. N. Y. (Geneva) Agr. Exp. Sta. Bul. 251, p. 177–194. 1904.

1919, both of which had been imported during the previous winter. These consisted of one lot (1) of Hollander variety, and one lot (2) of Copenhagen Market. Every field examined where seed from either of these two lots had been used showed more or less black rot.

A similar condition was found in the vicinity of Appleton, Wisconsin, where one dealer distributed a lot of imported seed of the Hollander variety (lot 3). In the two fields examined where seed from this lot had been used, black rot was very prevalent. Moreover in both instances plants from another lot of seed which had been used alongside the imported strain were practically free from disease except in rows immediately adjacent to the latter, where recent spread had occurred.

TABLE 1

*Summary of field observations on the occurrence of black rot in the Racine, and Appleton, Wisconsin, districts in 1919*

| LOCALITY | FIELD NUMBER | VARIETY | SEED LOT NUMBER | DATE OF OBSERVATION | PER CENT OF PLANTS INFECTED WITH BLACK ROT | PER CENT PREVENTED FROM HEADING BY BLACK ROT |
|---|---|---|---|---|---|---|
| Racine | 1 | Copenhagen | 2 | August 14 | 95 | 60 |
| | 2 | Copenhagen* | 2 | August 29 | 1 | |
| | 3 | Hollander | 1 | September 1 | 100 | 94 |
| | 4 | Hollander | 1 | September 1 | 59 | |
| | 5 | Hollander | 1 | October 13 | 95 | |
| | 6 | Hollander* | 1 | September 1 | 12 | 1 |
| | 7 | Hollander* | 1 | October 14 | 1 | 0 |
| Appleton | 8 | Hollander | 3 | October 10 | 74 | 13 |
| | 9 | Hollander | 3 | October 10 | 60 | 23 |

* Seed treated before planting with 1:1000 mercuric chloride for thirty minutes.

In several instances both in the Racine and Appleton sections, plants of the infected and the uninfected strains had been grown in the same seed bed, thus confirming the assumption that the pathogen had been introduced with the seed rather than by way of the soil. A summary of the observations in various fields is given in table 1. Spread of the disease from infected portions of fields to healthy plants of different origin in adjoining areas became very apparent several weeks after transplanting. In the case of field 3, for instance, counts were made in an adjacent plot of cabbage grown from clean seed. On September 1, eight to nine weeks after transplanting, the row next to the plot of infected Hollander showed 98 per cent of the plants with marginal infections of fairly recent origin; the second row showed 67 per cent; the sixth row 66 per cent; and the twelfth row 18 per cent. It is thus clearly evident that the black rot organism is readily disseminated in the field by natural agencies. In field 3 the disease progressed so rapidly that most of the plants were prevented from heading. In other fields where a large percentage of the plants headed, the soft rot which followed did considerable damage before harvest or caused heavy losses in transit.

It should be noted that in the three cases where the seed was treated before sowing with mercuric chloride, according to directions sent out by the Wisconsin Experiment Station, the disease was well controlled.

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## RESULTS OF WHITE PINE BLISTER RUST CONTROL IN 1919

S. B. DETWILER

Blister rust has made steady progress in the generally infected white pine region of New England and New York. Extensive areas of severely infected pines exist in most of these States. In a 41-acre pine lot at Littleton, New Hampshire, containing a total of 5442 native white pines averaging 6 inches in diameter, breast height, 55.4 per cent of the trees were recently found infected with the rust. This infection is entirely due to wild Ribes (currants and gooseberries). Wild gooseberries scattered over the 41-acre plot average 30 bushes per acre, 199 feet of leaf-bearing Ribes stems per acre. Strip surveys on compass lines a rod wide, totalling $22\frac{5}{8}$ miles in length, were run out from this pine lot in five directions across the surrounding country. Out of 2856 pine trees growing on this strip, 25.4 per cent were infected.

These data indicate that one-fourth of the pines in an area approximately 9 miles long and 8 miles wide are already infected with the disease. The oldest blister rust canker found on pine in this region is estimated to have started in 1906. These figures show that pine infection is progressing more rapidly than was estimated previously.

Control of the disease through destruction of currant and gooseberry bushes has been tried experimentally since 1916, in cooperation with the New England States and New York. The results in 1919 show that such control is practical and effective in protecting a given area of pines. On four control areas from which currant and gooseberry bushes were removed in 1916 and early in 1917, careful scouting this year failed to reveal any new pine infections occurring since the Ribes were destroyed. Under competent direction, unskilled laborers can be quickly trained to find and destroy over 95 per cent of the wild currant and gooseberry bushes the first time they go over the control area. In 1919, accurate checks were made on 1070 check plots containing over 750,000 bushes, to determine the thoroughness with which Ribes eradication was done. As part of their training, the crews made 642 checks, in which a total of 246,254 bushes, or 96.4 per cent, were uprooted in the first working, 8095 the second time over the ground, 905 the third time, 125 the fourth time, and 10 the fifth time. On 6 areas worked in previous years, totalling 1725 acres, 152,942 bushes were found in the original working and only 6474 bushes were found in checking. This is an efficiency rating of 95.9 per cent for the first working. Of the bushes found in checking, 4105 (or 2.6 per cent of the total found) were obviously missed when the areas were first worked; and 2369 (or 1.5 per cent of the total found) were sprouts and seedlings that had developed after the first working. On an acre basis, 89 Ribes bushes were destroyed in the first working, 2 bushes were missed, and 1 new plant appeared after the work was done. On 65 plots where the number of bushes was counted before and after the crews worked the areas, and where the men did not know their work was being checked, they found and destroyed 96 per cent of the bushes. Federal and state inspectors made numerous other checks which also show that the crews destroy in excess of 95 per cent of the bushes in the regular course of their work. Some of the check plots were gone over 10 to 15 times in order to be certain that no plants were missed.

It was found that on an area where only 87 per cent of the Ribes bushes were taken out in the first working, these bushes bore 96.6 per cent of the leaves. In this test, the area was gone over four times, and the leaves on 2194 bushes were counted. In the first working, 94,429 leaves were destroyed, and in the other three workings, only 3094 leaves were found. Measurements of the annual growth of 836 wild gooseberry bushes gave an average height growth of 5 inches per year for four years. Other counts showed that less than 5 per cent of the wild gooseberry bushes produce fruit, even in favorable crop years. Intensive study of Ribes seedlings on small plots demonstrate that they summer kill as well as winter kill, so that only a very small percentage live through the first year.

The cost per acre of Ribes eradication is low. The State crews eradicated 4,265,000 Ribes from 241,590 acres in 1919, at an average cost of 54 cents per acre. In 1918 the average cost was 66 cents per acre on areas totalling 135,239 acres. This reduction in cost was secured through scientific methods of locating Ribes in regions where they are not generally distributed. By this means, the average cost per acre in New Hampshire was reduced from 39 cents last year to 21 cents this year. In Rhode

Island this method reduced the cost from 27 cents per acre in 1918 to 11 cents per acre in 1919. The cost per acre in New York was much greater, due to an average of 92 wild currant and gooseberry bushes per acre, whereas in New Hampshire the average was only 10 bushes per acre. It is confidently expected that further experimental work will result in greatly reducing the cost without decreasing the efficiency of the work. In this connection, three years' experimental work in spraying Ribes with various chemicals indicates that dip oil and fuel oil, alone or in combination, are effective in destroying Ribes. Where the bushes are very numerous, the cost of eradication by chemicals is apparently much less than by hand pulling.

About $10,000 for coöperative Ribes eradication in New York in 1919 was subscribed by individuals and associations. In New Hampshire, 53 towns appropriated a total of $8514 for coöperative eradication of Ribes, and 34 individuals and firms subscribed an additional $2053. In Massachusetts, local coöperators furnished $1075. The sympathetic attitude of the public toward blister rust control is further evidenced by the fact that in New Hampshire 21,171 cultivated currant and gooseberry bushes were destroyed, and that compensation, as provided by law, was asked for by only three owners.

The necessity for developing local control of the blister rust is apparent from the rate of increase of the disease in native pine stands; and the demonstrated practicability of Ribes eradication fully justifies pine owners in undertaking this work on a large scale. Studies conducted by the Office of Forest Pathology, Bureau of Plant Industry, on the distribution of sporidia and the distance that infection has actually occurred upon pines from known infected Ribes, indicate that the Ribes-free zone should be, under average conditions, 200 to 300 yards in width. Tests of the longevity of the sporidia showed that they remained viable for a period of less than ten minutes where the humidity was 90 per cent.

Recommendations relative to control measures are summarized in the following extract from the resolutions of the Fifth International Blister Rust Conference, held at Albany, New York, on December 8–9, 1919:

> The Fifth International Blister Rust Conference urgently recommends the complete destruction of all Ribes within a distance of 200 to 300 yards of native and planted pines in regions where blister rust is present or within dangerous proximity. Owing to wide variations in the distance that spores spread from Ribes to pine, due largely to the nature of the vegetation intervening between the Ribes and pine, and to weather conditions, it is impossible to establish a fixed width of Ribes-free zone which will be equally applicable to all localities; but it is the opinion of this Conference, based upon recent investigations, that a Ribes-free zone of 200 to 300 yards in width will be sufficient to insure the commercial growing of white pine under average conditions. It is also recommended that white pine should not be planted in the locality of large plantations of Ribes, especially cultivated black currants.

Scouting was continued in the Middle Western and Southeastern States where the white pine is native and planted. The disease was found only in single plantations in Michigan and New Jersey. An area of disease approximately 150 miles long and 100 miles wide was found to exist in Wisconsin and Minnesota. At a conference held at Saint Paul, Minnesota, in October, 1919, to which representatives of the Lake States and Canada were invited, the conclusion was reached that the policy in these two states should be local control of the disease on the same basis as in New England.

A number of violations of the Federal quarantine against shipment of Ribes and white pines into the Pacific Coast and Rocky Mountain States were discovered in 1919. These states have been well scouted for four years and no blister rust has been found; but unless nursery stock of five-needled pines and of Ribes from infected regions is absolutely excluded from the far West, it is practically certain that the blister rust will eventually be introduced there. The fact that 65 species of wild Ribes are native to western North America and constitute a large proportion of the undergrowth in much of the western white pine and sugar pine forests, makes it imperative that the blister rust be kept out of this region if humanly possible to do so.

BUREAU OF PLANT INDUSTRY
WASHINGTON, D. C.

# LITERATURE ON PLANT DISEASES[1]

COMPILED BY ALICE C. ATWOOD AND FLORENCE P. SMITH, BIBLIOGRAPHICAL ASSISTANTS, BUREAU OF PLANT INDUSTRY

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SAMUEL McCUTCHEN BAIN

PHYTOPATHOLOGY

VOLUME X NUMBER 4

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# SAMUEL McCUTCHEN BAIN

S. H. ESSARY

WITH PORTRAIT PLATE X

Samuel McCutchen Bain was born at Eagleville, Rutherford County, Tennessee, January 14, 1869, and died at Knoxville, Tennessee, January 30, 1919. He was reared on a farm and educated in the public and private schools of his neighborhood. He received the equivalent of a classical college course in Eagleville School, but his scientific education was obtained for the most part by independent study. He studied French and German under the instruction of Miss Evelyn Franklin, whom he married at Henderson, Tennessee, June 6, 1891.

Early in life he developed a love for the sciences, especially for botany. With the few books he had at his command, he commenced the study of the flora of his neighborhood. Before his graduation, he was asked to give courses in the sciences in Eagleville School, where he showed great proficiency and ingenuity in the construction and handling of apparatus in teaching chemistry and physics. In 1890 he was called to Union University at Jackson, Tennessee, to assist in the teaching of sciences and French. He soon took up the study of the flora of that section and the collecting of material for his "Plantae Tennessei Occidentalis." During a vacation in 1892, he took a portion of his collection to the University of Tennessee to compare it with the specimens in the University herbarium. Doctor F. Lamson-Scribner, who was at that time Professor of Botany in the University, was greatly impressed with his work, and asked him to return the following year (1893) as his assistant. A short time later Doctor Scribner left the University, and Professor Bain was made Instructor in Botany and Geology and Assistant Botanist in the Agricultural Experiment Station. He was advanced in position gradually and was made Professor of Botany in the University and Botanist in the Station in 1901. He continued his studies of the flora of the state, and added very materially to the University herbarium. In his Experiment

Station work he soon became interested in the preparation and use of fungicides. After several years of intense study and experimenting, he published in 1902 his bulletin on "The Action of Copper on Leaves," which gained for him wide notice among the agricultural workers of America and Europe.

In 1904 he was appointed Special Agent and Collaborator in Cotton Breeding Experiments in the boll weevil investigations of the U. S. Department of Agriculture. He devoted one-half his time to this work for a number of years, working in West Tennessee, Arkansas, and Texas. While he was actively engaged in this work, he brought out the well known "Trice Cotton," which is now grown very extensively in the South. He spent more than a year in studying the oil content of cotton seed, the results of which have never been published.

About the time of his appointment in the U. S. Department of Agriculture, he became interested in the selection of plants resistant to disease. He selected and propagated a strain of red clover resistant to anthracnose, a disease which had well nigh put an end to the growing of red clover in the State. This selection work had much to do with restoring the growing of red clover in Tennessee. He soon started on a comprehensive scale the selection of other crop plants resistant to specific diseases, but this work was greatly hindered by illness during the latter years of his life. Several projects along this line were unfinished at the time of his death.

Professor Bain was as successful a teacher as an investigator. He never sought after large classes; but he had the ability to draw about him a small band of deeply interested students, who were inspired by his earnestness and devotion to his subject, as well as by his cheerful and lovable disposition. No teacher ever had more loyal pupils. He believed in teaching Botany as a pure science, believing that the practical application of the subject would take care of itself. As he had never had a teacher of Botany himself, he developed his own peculiar methods of presenting the subject. Through his wide study and his own researches, he was able to give his students instructions fully up to the standards set by other institutions. Many of his pupils have been called to teaching and station positions in other institutions. He published a number of papers on botanical subjects, and there are several unfinished manuscripts among his files. Some years ago, he began a paper, "Southern Contributions to Natural History," which was never finished. He left a valuable collection of pictures of the plants of the Tennessee mountains, among which are a number of color photographs unexcelled for their beauty. He was a pioneer in the development and application of the autochrome process of color photography in this country.

As a man, Professor Bain was noted for his unfailing good humor and optimism. He was under all conditions polite, genial, kind, and good-natured. Even during the years of his ill health, he never lost the spirit of good cheer. He was always ready to offer council, and not afraid to ask advice of others. My personal recollections of him are very vivid. His teaching was mostly out of doors, in the woods and fields. He took his classes on many long trips around the country. During the long summer vacations, he spent his whole time collecting and studying the flora of West Tennessee.

Professor Bain was a Fellow of the American Association for the Advancement of Science, a member of the Association Internationale des Botanistes, (1900); Member auxiliare (1902), Associé libre (1912) de l'Academie Internationale de Geographie Botanique; member of the St. Louis Academy of Sciences; member Royal Society of Arts, (1911); Member Botanical Society of America; charter member of the American Phytopathological Society; charter member Tennessee Academy of Science, (1912), and President of same in 1916.

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# A COÖRDINATION OF OUR KNOWLEDGE OF INSECT TRANSMISSION IN PLANT AND ANIMAL DISEASES

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## FOREWORD

During the past few years the question of disease transmission by insects has assumed such gigantic proportions that it has seemed advisable to canvass the whole subject in a more or less general manner in order to determine whether certain principles may not be found applicable to insect transmission of both plant and animal diseases.

The literature of human and animal pathology is now full of examples of insect transmission of disease; and following the early work of Merton B. Waite (158, 1892) and Erwin F. Smith (125, 1896) the attention of plant pathologists has been more and more directed toward this phase of the subject. As a matter of fact, the whole question of insect transmission of disease may be stated in such a way as to cover both the plant and animal kingdoms in one statement. However, since this paper is written primarily in the interests of plant pathologists, only sufficient examples will be given among animal diseases to show the distinct correlation of insect transmission in these two branches of pathology.

## GENERAL RELATIONS OF INSECTS TO PARASITIC DISEASES

There are many conditions of disturbed function or anatomy caused directly by insects or other small animals without the intervention of parasitic microörganisms. Such, for instance, are the local irritations resulting from the bite of the tick, flea, spider, or mosquito, and the various dermatoses caused by lice, chigoe, red bug, and mites, among animals; and the various forms of erinose and stigmonose, and many insect galls, among plants. Some of these conditions may as truly be included under the heading of disease as disturbances caused by microörganisms, but they scarcely come within the scope of the present paper.

Setting aside, then, such cases of disease as are of direct insect causation, there are three general relations which an insect may bear toward diseases due to microörganisms. The infective principle (1) may be picked up by the insect on external parts of its body, such as the legs or outer mouth parts, where it may retain its virulence for a certain period of time.

In these cases the organism may be (a) directly inoculated, as into wounds made by the insect carrier, or (b) accidentally sown from its body under such conditions that infection follows. Such purely mechanical dissemination may be the means of spreading infection over a wide territory.

Again, (2) an insect without itself carrying infection, may cause wounds through which parasitic microorganisms find easy access into the plant or animal body, brought there by other agencies.

Furthermore, (3) microorganisms are often taken up by an insect during the feeding process and passed along unharmed through the alimentary tract. If the infective principle (a) is merely taken into the body of the insect, and, without change or multiplication, passed out in a normal way, this mode of dissemination may be spoken of as *mechanical internal transmission.* If, however, the infective principle (b) develops or multiplies within the insect body, or by its own movement goes counter to the usual course of substances through the body and finally arrives at a point from which it may be successfully transferred to its alternate host, the mode of dissemination is designated as *biological internal transmission.*

An insect may transmit a parasite to susceptible parts of the host without wounding it, and this may be done either by external, or by internal (mechanical or biological) carriage, or by both. In other cases it may also directly introduce the same parasite into wounds which it makes, and here again the infection may be carried either mechanically or biologically. With respect to the same disease there may be still other cases where individual insects, which are in no sense carriers, make wounds through which the parasite already present or subsequently present is introduced. In other words there is more or less overlapping of the different forms of insect transmission, but since the extremes are clear and definite and since one form of transmission or another generally predominates, this classification serves a useful purpose, and so far as the data at hand warrant, it will be followed in the present paper.

## EXTERNAL TRANSMISSION OF PARASITIC MICROORGANISMS

### *External dissemination and direct inoculation by the insect carrier*

Mechanical external carriage and inoculation of pathogenic microorganisms by insects is very common among both plant and animal diseases. By way of example among diseases of vertebrates it may be stated that some of the parasitic trypanosomes are mechanically carried in or on the probosces of blood-sucking insects after they have taken blood from an infected animal. When these insects feed a second time the trypanosomes pass directly into the vertebrate host, without having undergone any further change or development (cf. also p. 214).

*Bacterial diseases.* Among plants fire blight of pomaceous fruit trees caused by *Bacillus amylovorus* (Burrill) Trev. is the first disease of microbic origin shown by definite and extensive experimental evidence to be directly disseminated by insects. For the most part this disease is carried and definitely inoculated either into the fluid on the susceptible nectar discs or into wounds made by the insect carriers. Forbes in 1884 (45, p. 150) had suggested that probably tarnished plant bugs were often active agents in conveying the "virus" of the blight from tree to tree by inoculation with the infected sap. At this time, however, the question of insect transmission in diseases of both plants and animals was a rather moot point, and such speculative suggestions pro and con were the order of the day. It remained for the epoch-making studies of Waite (158–162, 1892–1906) to actually demonstrate the dependence of a plant disease on insect dissemination. He proved experimentally (158, 1892) that the bacillus of fire blight is carried from flower to flower by bees and other insects visiting the blossoms for pollen and nectar, and that the bacilli are present and multiply in the nectar after insect visits and that blossom blight is contracted in this manner and only in this manner. Later (159, p. 298, 1896) he states that the warm, moist weather which brings out the blossoms is particularly favorable to the development of the blight and that the bacteria-laden gum from "hold-over" cankers is then exuded copiously. Bees, wasps, and flies are attracted to this ooze and undoubtedly first carry the microbes to the blossoms in this manner. From these first infected flowers the disease spreads with great rapidity. It is almost certain also, he says, that after the blooming period insects carry the blight to the young, growing tips. The germs on the new growth of the season first appear on the nectar discs (160, 1898), where they live and multiply, and they can enter the host at this point without puncture or other injury. In 1902 (161) he stated, "We have scarcely been able to find a direct normal method of introduction of the disease to twigs without the introduction of some mechanical or insect puncture." Waite observed upwards of 40 species of insects visiting pear blossoms, and many of them were demonstrated experimentally to be carriers. The honey bee was shown to be most important in spreading the blossom blight. In different parts of the United States *Bacillus amylovorus* (Bur.) Trev. was repeatedly isolated from the mouth parts of this insect. Again and again the blight was inoculated inside of bags and kept in, and outside of bags and kept out. The direct insect relation was clearly established. It was found that insects not only distribute the blight but are active agents in puncturing the germs into the tissues (162, p. 143, 1906).

Following this pioneer work of Waite many investigators have reported upon various phases of the blight-insect relation; and up to the present time more investigators have worked upon the insect dissemination of this blight than upon that of any other plant disease. In 1909 Whetzel and Stewart (168, p. 39) concluded on observational evidence that aphids and leaf-hoppers are largely responsible for introducing the germ into the tips of growing shoots, while these insects, together with the curculio, frequently infect wounds which they make in the fruit. In the pear orchard they found the disease seldom appearing until the trees begin to bear, that is, until blossoms are present for inoculation by insects. According to the observations and experimental evidence in one locality given by D. H. Jones in 1909 and 1910 (68–69) it appeared that a large proportion of the new twig and water-sprout infections, and practically all that occurred after the blooming period, was due to dissemination and inoculation of the blight bacillus by aphids, chiefly of the two species *Aphis mali* Fitch and *Schizoneura lanigera* Hausm. Aphids were also found to be the principal means of spreading the disease in apple nurseries, and control of the aphids reduced the number of blight infections. D. H. Jones also traced many cases of blight in pear trees to wounds due to the bark-borer (*Scolytus rugulosus* Ratz.) and obtained cultures of the blight bacillus from several of these beetles taken from diseased pear trees. These beetles were also found in apparently healthy bark which later developed the blight around the vicinity of their borings. In 1911 (70–71) Jones gave further circumstantial evidence and finally by direct experiment proved that this beetle carries and inoculates the fire-blight germ. V. B. Stewart in 1913 (143–144) observed that fire blight is spread by aphids in quince nurseries, and showed by direct greenhouse experiments that infections occur through aphid punctures in young, succulent tissues. However, in the region under his observation, he found the tarnished plant bug (*Lygus pratensis* L.) to be much more important in the dissemination of the blight germ in nursery stock. By direct experiment he showed that these insects visit blighted tissues, become smeared with the gummy exudate, carry the bacteria to the tender growing tips, and inoculate the host tissues while sucking up the sap, with the result that the twigs soon develop typical blight. Referring to the collar-blight form of the disease on apple trees, C. R. Orton and Adams in 1914 (100) remark that "the apple-tree borer is probably the most active agent in the spread of the causal organism," and later (101, 1915) they found that in 90 per cent of the observations on collar blight, apple-tree borers had been associated with the disease at the collar. Whether this insect was a direct carrier or only provided points of entry for the blight germ was not determined. However, on account of the almost universal presence of wooly aphis (*Schizoneura lanigera* Hausm.) in collar-blight cases in Pennsylvania, it was thought possible that the aphis might be a carrier to the wounds made by borers. These aphids congregate wherever exudations of sap occur. In explanation of an unusual outbreak of apple blossom-blight, Reed, in 1914 (111), attributed the large numbers of insects, particularly honey bees, during the blooming period to the preceding mild winter and late spring. He considered this particular epidemic as due to the concomitance of large numbers of blossoms, insects and hold-over cankers. Stewart and Leonard in 1915 (145) proved by direct experiment on apple seedlings that four sucking insects (*Campylomma verbasci* Herrich-Schaeffer, *Orthotylus flavosparsus* Sahlberg, *Poeciloscytus basalis* Reuter, and *Adelphocoris rapidus* Say.) are capable of disseminating and inoculating the blight germ. These authors consider the tarnished plant bug (*Lygus pratensis* L.) and *Campylomma verbasci* particularly important because of their abundance in nurseries and because they attack chiefly the tips of shoots where conditions are best for infection. The apple red bugs and the false tarnished plant bug are also given on circumstantial evidence as probable disseminators. In Wisconsin, A. C. Burrill experimentally demonstrated (22, 1915) that aphids (*Aphis avenae* Fab.) and leafhoppers (*Empoasca mali* Le Baron) carry infection and inoculate it into the wild crabapple (*Pyrus coronaria* L.). As a result of preliminary studies, Heald (57, 1915) concludes that leaf invasions by *Bacillus amylovorus* through water pores or stomata may in some cases be of common occurrence. (Up to this time the general supposition, based upon much careful work, had been that with the exception of the nectary infections, mechanical injuries of some sort were necessary for entrance of the organism. If this method of infection is of general occurrence and if it is shown that the germ can work its way from leaf to twig, the control of the disease will be greatly complicated.) Gossard (51), and Gossard and Walton (52) in 1916 showed experimentally that *Bacillus amylovorus* may remain viable for several days in honey and in the honey dew of aphids. These facts increase the sources of possible insect contamination. In further insect experiments (1916) Stewart and Leonard (146) found that the apple leafhopper (*Empoasca mali* Le Baron) and a capsid (*Plagiognathus politus* Uhler) gave infections, while two species of fly (*Pollenia rudis* Fabricus, and *Sapromyza bispina* Loew) and the common pear psylla (*Psylla pyricola* Förster) gave negative results. They state that possibly flies may carry infection to blossoms and wounds, but on account of their method of feeding they think it is unlikely that these insects inoculate the bacillus directly into twigs. Merrill observed a direct relation between aphid infestation and amount of fireblight infection (90, 1915) and later (91, 1917) demonstrated that aphids as readily deposit their eggs in blight cankers as in other rough places in the bark, and soon after hatching from the eggs in spring they crawl through the blight exudates. He also obtained infections by caging contaminated aphids on apple twigs (cf. p. 200).

In 1896 Erwin F. Smith (125, p. 22) experimentally transmitted the bacterial *wilt of solanaceous plants* caused by *Bacterium solanacearum* Sm. using the Colorado potato beetle, *Leptinotarsa decemlineata* Say (formerly *Doryphora decemlineata* Say), previously fed upon diseased shoots.

In the year following (126, p. 409, 1897) E. F. Smith reports infection of cabbages with the black rot organism, *Bacterium campestre* (Pam.) Erw. Sm., by means of larvae of the American cabbage worm, *Autographa brassicae* Riley (formerly *Plusia brassicae* Riley), previously fed upon diseased plants. He also considered it almost certain that the disease may be transmitted by various bugs, beetles, and insect larvae that feed on cruciferous plants. Water-pore infections were later (126, 1897; 128; 129, v. 2, p. 306, 1911) found to offer the usual mode of entrance for the bacteria into the host tissues, but it was shown experimentally that infections also occur through the gnawings of insects and slugs, and that these small animals may be the means of spreading infection to the water pores. Substantially these results were confirmed by Russell and Harding in Wisconsin (114, 1898). Brenner in 1904 (19) confirmed the results of Smith and of Russell and demonstrated that aphids also successfully disseminate and inoculate the black rot bacteria into the host. He found that a single plant louse was sufficient to transmit the disease.

The experiments by Manns (83, p. 111, 116, 1909) show that a bacterial *blade blight of oats* is distributed and inoculated by two species of aphids, *Macrosiphum granaria* Buck, and *Syphocoryne avenae* Fabr.

Burger (21, 1914), working with a bacterial rot of lettuce, found large numbers of reddish-brown aphids present in infected fields, and showed by careful experiments that these insects could distribute and inoculate the disease.

*Fungous diseases.* Sturgis (150, p. 191, 1898) reports a direct relation between bees and primary pod infections with lima-bean mildew, caused by *Phytophthora phaseoli* Thaxt. He discusses the special adaptation of the flower for insect cross pollination, in which process the base of the ovary and the style are momentarily exposed and come in contact with the body of the bee. This insures cross pollination, but if the bee's head or abdomen has previously touched a mildewed pod, fungous infection is no less certain to follow. Young pods often show evidence that infection has already occurred before the fall of the blossoms and that it always has taken place at the extreme base or tip of the young pods. These, he says, are the exact points touched by bees in visiting the flowers.

Waite (161, 1902) thinks from extended field observations that bees are largely responsible for the rapid spread of peach and plum brown rot caused by *Sclerotinia fructigena* (Pers.) Nort. Wasps and soldier bugs and several other puncturing insects are also concerned in making openings in sound fruit. Bees rapidly follow in the punctures made by other insects, and in California during June and July hundreds and thousands of bees may be seen swarming over the early ripening peaches and Japanese plums. In going over decayed fruit he thinks that they carry the fungous spores into wounds made by other insects, and states that they even eat large holes in the fruit, thus probably carrying infection and also inoculating it directly into the fruit. W. M. Scott and Ayers in 1910 (119, p. 13) state that sucking insects of the squash-bug family (Coreidae) have been observed to puncture healthy and diseased fruits indiscriminately, thus not only distributing the spores but probably also inserting them into the peach. But these investigators and also Scott and Quaintance (120, p. 3–4, 1910) consider the plum curculio by far the worst offender. In the feeding and egg-laying processes the fruit is punctured and these wounds form a nidus for the brown-rot spores. This insect punctures the fruit through the coating of spray and is almost certainly also a carrier of the spores, so that the control of brown-rot is thus greatly complicated.

James Scott in 1907 (118) states that certain insects, and among them the fungus fairy fly (*Caecilius flavidus* Steph.), are to be regarded as secondary causes of the *larch canker*, since the causal fungus gains entrance through their injuries to the tender bark. Large numbers of the fairy fly and its larva, covered with spores, are found on the cankers and in the "witches brooms" and so may distribute infection to fresh districts. The tiny punctures of the larch aphid (Chermes) are also known to be a predisposing cause of this disease and should be further investigated. The following year Maxwell (88) reports that the causal fungus gains entrance through wounds, but he considers that impaired vitality from any cause has more to do with predisposition to this disease than any direct connection between aphids and infection with the causal fungus, *Dasyscypha calycina* Fckl.

The *bud-rot of carnations* was first reported by Wolcott in 1905 (170), and on account of the constant presence of mites was then attributed entirely to their injuries. In 1908 Heald (56) proved that the disease is due to the fungus, *Sporotrichum anthophilum* Peck, and from observations and experiments he considered it almost certain that the mite acts as a carrier of the fungous spores which it inoculates into buds visited for feeding purposes. Wolcott in 1908 (171, p. 31) found this mite to belong to a new species which he named *Pediculoides dianthophilus*. During the same year Stewart and Hodgkiss (142, p. 112–115) obtained substantially the same results. They add, "The fact that the disease sometimes destroys the interior of buds before they have begun to open is strong evidence that the spores are carried there by some animal and mites are the only animals found constantly in diseased buds." The mites probably visit the healthy buds to feed on floral tissues. The spores carried on their bodies germinate, attack the interior of the buds through injuries, and later cause the decay. In the decayed tissues the mite finds a congenial breeding place from which the young later migrate to healthy buds, thus completing the cycle.

In Holland, Ritzema Bos (112, 1906), and Quanjer (105, 1907) working with *black leg* of *cabbage* (caused by *Phoma oleracea* Sacc.) showed experimentally that infection enters through wounds made by insects, particularly the cabbage maggot, *Pegomya brassicae* Bouché (formerly *Anthomyia brassicae*), and through injuries by wire worms and snails. They state that apparently all these animals also transfer the fungus from diseased to healthy plants. Manns (84–85, 1911), working in America, did not find the association of maggots and wire worms to be as important as did these European investigators. However, he considered these animals, together with the cabbage curculio, to be quite generally instrumental in to some degree aiding the fungus in its distribution and attack.

From his personal observations and correspondence with other countries relative to *Nectria ditissima* Tul., Ducloux (38, 1910) concluded that wooly aphis is in some way associated with the spread of this *apple canker*. Wiltshire, in 1914 (169) refers to the current opinion associating this insect with outbreaks of the Nectria canker, and states that Descours Desarces has proven that these insects carry the conidia on their wooly covering. Wiltshire, however, obtained negative results by inoculations made in various ways upon fresh aphis injuries. In late summer, after the bark bursts around these injuries, he thinks that entrance may possibly be afforded, and this conclusion is supported by the fact that canker attacks usually occur in the year following outbreaks of wooly aphis. Massee (87), in 1915, stated that the rapid spread of the Nectria canker coincided with the introduction and spread of the wooly aphis (*Schizoneura lanigera* Hausm ) into England. Its injuries on the branches of apple trees are exactly of the nature required by the fungus for entry into the host tissues. These insects, also, he says, carry the fungous spores.

In connection with the *pineapple fruit-rot* and *leaf-spot* caused by the fungus, *Thielaviopsis paradoxa* von Hoh, (formerly T. *ethaceticus* Went.), Larsen (75, p. 26, 1910) says that insects aid the fungus by injuring the fruit and leaf surfaces and by distributing the spores. The mealy bug (*Pseudococcus bromeliae* Bouché), and a grasshopper (*Xyphidium varipenne* Swezey) are considered most important in this connection. Of the leaf spots caused by this fungus, 75 per cent were found to have originated through grasshopper wounds. Ashby (7, 1917) considers on circumstantial evidence that the spores of this same fungus, causing one of the "leaf-bitten" diseases of pineapple, are spread by the wind, by wind-borne raindrops and by insects. He says further that the striped weevil, *Metamasius sericeus* Oliv., which is attracted to rotting patches on the heart leaves, is an active flier and undoubtedly plays a part in disseminating the spores. (cf. p. 201).

Grossenbacher and Duggar (55, 1911), on the basis of circumstantial evidence, consider it probable that various insects, and especially a small beetle, the American currant borer (*Psenocerus supernotatus* Say.), are instrumental in spreading and inoculating the spores of *Botrysphaeria ribis* Gross. & Dug., the cause of a *cane blight of currants*.

Taubenhaus (151, 1912) considers from circumstantial evidence that aphids and red spider distribute and inoculate spores of the anthracnose of *sweet peas*.

In 1915 Nowell (95) associated the *internal disease of cotton bolls* with the attacks of certain sucking insects. This disease is characterized by a discoloration of the lint in unopened bolls without external signs of injury. In one series of observations Nowell and his associates found (a) the disease absent in the absence of the cotton stainer bug (Dysdercus spp.), (b) a high percentage of the disease where the plants were infested with this insect, (c) a constant association of cell proliferations within the diseased carpels, suggesting injury by a piercing insect, and (d), where the stain was just beginning to show, the occurrence of water-soaked spots suggesting insect punctures. Punctures with a sterile needle usually gave nothing but mechanical injury. Cage experiments with cotton stainers, on the other hand, gave a high percentage of disease, while the controls gave little or none. It was thought probable that the internal boll disease is caused by a microörganism brought in by this bug. The following year Nowell (96) discovered a specific fungus causing the disease and showed that it was carried by or followed closely upon the attacks of plant feeding bugs. In his studies of 1917 (97–98) infection was found to take place without apparent exception through bug punctures, and mainly through those made by the cotton stainer. Control measures against this insect reduced the disease to negligible proportions over large areas. In 1918 (98) several other fungi were found also capable of causing internal boll disease, and these specific fungi were isolated from the fruits of local plants belonging to seven different orders. Careful cage experiments demonstrated that the punctures of the cotton stainer, green bug (*Nezara viridula* Linn.), the leaf-footed tomato bug (*Leptoglossus balteatus* Linn.), and the red tomato bug (*Phthia picta* Drury) may all serve as channels for infection. Not a single case was found of infection other than through a bug puncture, and strong evidence was given that bugs are also the carriers. It appears that stainers carry one type of fungus and green bugs another, and this was correlated with the presence of these specific fungi on the respective preferred food plants of these two insects. The species of fungus causing the disease in any one case depended upon the preceding food plant used, but where bugs were taken from healthy plants no infection followed. The pea chink (*Edessa meditabunda* Fabr.) always gave negative results whether previously fed upon healthy or diseased plants, (but this insect scarcely pierces the outer covering of the bolls).

Turner (157, 1918) found an association between kernel spot of pecan and the green soldier bug, *Nezara viridula* Linn. In 1916 he observed severe infestation of these green bugs in pecan groves, followed by a severe attack of kernel spot. Accordingly the following year some of these insects, collected from cowpeas, were enclosed for several weeks with pecan nuts on the trees. Examination of the nuts after they had ripened showed every one to be affected with kernel spot, while only two or three cases were found among several hundred nuts on the same tree, not confined with these insects. He had not determined upon the presence or absence of the fungus (*Coniothyrium caryogenum* R.) described by Rand in his preliminary studies upon this disease (105 a, p. 330), but he considered, in any case, that *Nezara viridula* is an important agent, either as a direct cause or as a means of dissemination.

### *External dissemination and accidental infection without direct inoculation*

*General discussion.* In many other instances the relation of the insect is predominently that of a mechanical carrier only. In typical cases of this type of dissemination the infective principle is not directly inoculated into the host but accidentally sown from the insect's body, as e.g. on wounds or other portions of the host favorable for infection. This method of transmission is common in diseases of both animals and plants. In studies of human and animal diseases it has been shown repeatedly that many insects which come in contact with infected material, such as fecal matter, pick up large numbers of disease germs on their legs and bodies and carry these germs great distances, depositing them again on fecal matter, or more important still, on food or wounds or sensitive membranes. The house fly especially has been convicted of the external transmission of many species of bacteria. Most of the flies which frequent houses and food or visit man because of attraction to bodily secretions or injuries are also attracted to and breed in excreta or garbage. Hence the contamination of food by direct transportation from infected excreta is a very simple matter.

*Bacterial diseases.* By way of examples in animal pathology *typhoid fever* and *cholera* may be mentioned. The typhoid bacilli are often taken up from infected material by the feet or mouth parts of flies and deposited upon liquid or solid food by means of which access is easily obtained to the human alimentary tract. Occasionally flies carry infection internally for long periods of time (cf. p. 33).

Cholera vibrios are also rather commonly disseminated by flies, particularly in those countries like India where dejecta are not properly disposed of.

Among bacterial diseases of plants, fire blight of pomaceous fruit trees is frequently contracted through various injuries to the young shoots or tender, fleshy bark. In these cases insects and birds are the agents of distribution (Waite, 160, 1898). Whetzel (166, p. 115, 1906) observed that flies constantly followed the pruner for the exuding sap and thought they might be agents in transferring the bacilli in this way from diseased to healthy trees. Hotson (62, 1916) found blight starting in hail injuries on twigs and fruit and considered the rapid spread to uninfected trees as probably due to various kinds of insects, and especially ants which he found attracted by the exuding sap (cf. p. 192).

R. E. Smith, C. O. Smith and H. J. Ramsey (134, 1912) observed flies on diseased walnuts affected with the blight organism, *Pseudomonas juglandis* Pierce, probably attracted there by the exuding organic matter mixed with bacteria. The blight organism was isolated from the bodies of such flies.

*Fungous diseases.* Magnus (82, p. 95, 1891) thought the teleutospores of species of Diorchidium well adapted to insect distribution because of the fact that they are sticky and are set free in dew drops or other moisture. Klebahn (73, p. 19–20, 1904) considered that insects play an important role in the distribution of *rusts* in general, and also suggested that the bright color of some species might attract insects; and Falck (42, p. 56, 1904) thought, on account of the odor frequently emanating from affected parts of the host, that the pycnospores were probably distributed by insects. Freeman in 1905 (46, p. 29) says that the early spring spores of rusts are often accompanied by structures [aecidia] exuding sweet fluids attractive to insects, which may assist in sowing the spores. Johnson (66, 1911) in his work on floret sterility of wheats in the southwest found this condition to a large extent caused by rusts. Examination of large numbers of thrips showed rust spores attached to the appendages of a considerable proportion. As the thrips were very numerous and very active, he considered that large quantities of spores must be distributed over individual plants and between fields. The prevalence of rust spores between the glumes of wheat florets and the consequent rusting of the heads was thus tentatively accounted for.

Ludwig (79, 1892) calls attention to the fact that those *smut* spores which have a roughened cell wall are found on those parts of their hosts which are regularly visited by insects, while the smooth spored sorts occur on wind pollinated plants or on parts not regularly visited by insects. Brefeld and Falck (18, 1905) and later Jensen (65, 1918) suggest that in anther smut of plants pollinated by insects as in *Melandryum album* Garcke and *Saponaria officinalis* Linn. the smut spores are not disseminated like loose smut spores, by the wind. The flowers are visited by insects and in the case of Melandryum especially by night-flying butterflies. These insects stick their probosces into the blossoms to get honey and in this way the spores are forced out, and getting onto the insect body are said to be thus distributed to other flowers.

According to Clinton (31, 1902) insects, and especially the small pomaceous flies of the genus Drosophila, are undoubtedly one of the chief agents in the dispersal of *bitter-rot of apples* caused by *Glomerulla cingulata* (Stonem.) S. and v. Schrenk. The Drosophila flies breed abundantly in the decaying apples; and, in a damp chamber experiment, infection was carried by these flies from bitter-rot to sound apples. Thomas J. Burrill (23, 1907) thought that the spores were to some extent distributed by pomace flies but considered that no insects are largely instrumental in the distribution or development of this disease.

Cobb (33, p. 116, 1906) thinks that the pineapple disease of sugar cane caused by *Thielaviopsis paradoxa* von Hoh. (formerly *T. ethaceticus*) is often assisted in its distribution by insects, and that in all probability they are attracted by the odor. Diseased cuttings are often infested with mites and with the larvae of small beetles of the genus Carpophilus. Examination of these beetles repeatedly disclosed spores of the fungus on their bodies, and as they move from cutting to cutting, he thinks that they undoubtedly spread the disease. These beetles have a grinding crop with teeth fine enough to destroy the spores, only fragments of which were found in their intestines. Larsen (75, p. 26, 1910), working with the same fungus causing a fruit rot of pineapples, says that the fruit beetle (*Carpophilus humeralis* Fabr.) does not rupture the surface so far as could be ascertained, but that it distributes the spores from affected pineapples to newly made wounds in healthy fruits. The vinegar fly (*Drosophila ampelophila* Loew.) and other flies are also said to be important factors in distributing the spores (cf. p. 198).

That the *chestnut blight* fungus, *Endothia parasitica* (Murr.) P. J. & H. W. And., may be distributed by insects was early inferred by Murrill (94, 1906), Hodson (59, 1908), Metcalf and Collins (93, 1911), Metcalf (92, 1913), Fulton (47, 1912), and Rankin (110, 1912). Craighead, in 1912 (35) found by cultural methods that the spores of this fungus are not viable after passage through the alimentary tract of the longicorn beetle, *Leptostylus macula* Say. This result was later confirmed by Anderson and Babcock (6, 1913), but they succeeded in culturing the blight fungus in some cases from the bodies of ants artificially contaminated or collected from pustules on the trees. Studhalter (147, 1914) by microscopical and cultural tests found twenty species of insects carrying Endothia spores on their bodies. The longicorn beetle was found to carry the largest numbers of any of the insects tested, showing that even if the spores do not survive intestinal passage through this insect it may still be a factor in the dissemination of the fungus. Later in the same year Heald and Gardner (58) showed that the pycnospores retained their viability for some time in the soil, and suggested that this offers a possible means of transport of the spores in dried mud on insects, birds, squirrels etc. Studhalter and Heald (148, 1915) found that the largest number of pycnospores were obtained from birds and insects within about two to four days after a rain. This corresponded closely with the time the largest numbers of spores were obtained from healthy bark below chestnut blight lesions. About the same time Studhalter and Ruggles (149), reviewing their experiments and observations relative to insect transmission of this disease, conclude that some insects carry a large number of spores of *Endothia parasitica* and that they are important agents in the local dissemination of the disease. This, they state, is especially true of the longicorn beetle. On the other hand, Craighead (36, 1916) suggests that in order to play an important part in dissemination, an insect must migrate from diseased to healthy trees. Most of the Coleoptera discussed by Heald and by Ruggles he says do not have this as a normal habit; the longicorn beetle never frequents healthy trees and most of the insects discussed by these authors are only occasional visitors. With respect to the chestnut blight, Craighead considers that the important role of insects consists in providing fresh wounds for the entrance of the chestnut blight fungus (cf. p. 204).

L. R. Jones, Giddings, and Lutman (72, p. 40, 1912) assert that washing and dashing about by rain is the most effective agency for local dissemination of the potato fungus, *Phytophthora infestans* (Mont.) de Bary. For longer distances wind and insects probably coöperate. Larvae of the Colorado potato beetle previously fed on blighted leaves were kept in a damp chamber over night on two healthy potato plants both of which developed the disease in six days. The mature beetles are, of course, capable of carrying the spores to greater distances and are doubtless important agents in the sowing of late blight infection in new fields

Taubenhaus (152, 1913) claims that the *black rot* of *sweet potato* caused by *Sphaeronema fimbriata* (E. & H.) Sacc. is spread in storage by means of mites and spiders. The disease followed when mites and spiders were transferred from diseased to healthy roots and pure cultures of the causal fungus were obtained when they were allowed to crawl over poured plates of solid culture media.

The same investigator (154, 1914) states that fruit flies are constantly associated with *soft rot* and *ring rot* of *sweet potatoes* in storage.

Whetzel (167, 1915) states that the spores of Botrytis blight of peony caused by *Botrytis vulgaris* Fries and *Botrytis cinerea* Pers. are produced in great quantities and are carried by wind and insects. Ants seem to carry the spores from the base of diseased stalks to the buds of healthy plants. Here in the exuded sugary solution so abundant on the unopened buds, he says, the spores find ideal conditions to germinate.

McKay and Pool (81, 1918) obtained *Cercospora beticola* Sacc. in culture from the "yellow bear" (*Diacrisia virginica* Fab.) and in one case from the greater sugar-beet leaf-beetle (*Monoxia puncticollis* Say.) but not from the sugar-beet webworm (*Loxostege sticticalis* Linn.). These insects were collected in diseased beet fields and show that the spores of this fungus may be carried on the bodies of some insects.

In work conducted at the Bureau of Entomology Field Laboratory at Thomasville, Georgia, in coöperation with the State Board of Entomology of Georgia, the late C. G. Crittenden and G. D. Smith proved that the green bug (*Nezara viridula* Linn.) after walking over a boll affected with *cotton anthracnose*, would leave a culture of the causal fungus in each foot print (Reported by Pierce).

## INFECTION THROUGH INSECT WOUNDS WITHOUT DISSEMINATION BY THE WOUNDING AGENT

Many instances are on record, particularly among plant diseases, where insects are not directly concerned in transmitting the causal microörganisms, but where injuries which they initiate afford the necessary openings into the host body through which infections later take place.

*Bacterial diseases.* Edgerton (39, 1912), working with angular leaf-spot of cotton caused by *Bacterium malvacearum* Erw. Sm., states that there are a number of insects that puncture the bolls and thus aid the bacteria in gaining entrance. A considerable percentage of the water-soaked initial infections, especially in the older bolls, have these small punctures at the center. Faulwetter (43, 1917; 44, 1919) confirmed these observations and at the same time by cage experiments with jassids, found the part played by these insects in direct transport of the bacteria to be negligible.

In the disease of sugar-beet and nasturtium leaves caused by *Bacterium aptatum* Br. & Jam. Brown and Jamieson (20, 1913) found from inoculation experiments that infection takes place only through bruises or wounds due to insects or other mechanical causes.

*Fungous diseases.* Barber (13, 1901) reports that the red rot of sugarcane caused by *Colletotrichum falcatum* Went. appears in some localities to require a borer hole for entrance and that it usually does not spread to other parts of the cane from the joint thus entered. In other cases it is propagated by use of diseased cuttings. Lewton-Brain (78, 1908) states that the fungus is essentially a wound parasite and that a deep wound reaching the soft inner tissues is necessary for its entrance. Injuries produced by the Hawaiian sugar-cane borer (*Sphenophorus obscurus* Boisd.) are the only insect wounds in Hawaii answering this purpose, those made by leaf hoppers not being deep enough. Again, Butler (24, 1906), and Butler and Hafiz Kahn (25, 1913) claim that uninjured canes are not attacked unless the parent stem is affected. The fungus is common around injuries made by a minute borer in the midribs of the leaves, and around wounds caused by stem borers.

In discussing onion blight caused by the fungus *Peronospora schleideniana* de Bary, Whetzel (165, p. 145–146, 1904) says that leaves which have reached maturity or those weakened by breaking or by ravages of thrips seemed most easily infected by the fungus, while maggots also contribute their share in predisposing to this disease.

During experiments with biologic forms of *Erysiphe graminis* DC, Salmon (115, 1905) found that one of the brome grasses (*Bromus racemosus* L.) was immune against one form of this powdery mildew when its leaves were uninjured but became susceptible when they were punctured by aphids.

Lewton-Brain (77, 1907) speaks of the Melanconium fungus of *rind disease of sugar cane* as a wound parasite which in many cases probably enters through leafhopper punctures, but he considers the lowering of the host vitality by repeated attacks of this insect as more important in predisposing to infection. It is claimed by Cobb (34, p. 107, 1909) that entrance to this fungus is afforded through tunnels of stem borers and through punctures of leaf hoppers in the tender upper internodes.

Murrill (94, 1906) suggests that green fly and twig borer wound the tree and thus give entrance to the chestnut-blight fungus (*Endothia parasitica* (Murr.) P. J. & H. W. And. Ruggles (113, 1914) states that the making of wounds in trees, which open the way for infection, is probably the most serious part which insects have in the dissemination of this disease. Among the insects most important in making wounds are 17-year cicadas, tree hoppers, bark borers, and bast miners. A careful study of cicada wounds in eastern Pennsylvania showed 86 to 94 per cent of all infections to be in cicada stings, and observations and experiments seemed to show that the bast miner also is responsible for much of this class of infection. A later report by Studhalter and Ruggles (149, 1915) further incriminates the bast miner as an important factor in chestnut blight infection, on account of the large number of openings which it makes in the bark. Craighead (36, 1916) after a critical review of the work of Heald and of Ruggles on insect dissemination of the chestnut bark disease, asserts that no direct insect relation has been shown to exist in this disease such as is the case in pear blight and ergot. The rôle of insects, he says, is probably insignificant in the dissemination of this disease, but their rôle in the making of wounds affording entrance to the fungus is far more important. The longhorned beetle (*Leptura nitens* Forst.), the chestnut bast-miner (*Ectoedemia phleophaga* Busck) and also three species of Sesia are mentioned as of importance in providing fresh wounds in living, healthy trees for entrance of the fungus causing chestnut bark disease (cf. p. 201).

With regard to pecan scab caused by the fungus *Fusicladium effusum* Wint., Waite (163, 1911) says: "An interesting feature was found in relation to the life history of the disease, namely, that a plant louse, which becomes common on the pecan in May, punctures the epidermis in a regular way along the veins and veinlets. The punctures of this plant louse are used as points of entrance by Fusicladium."

## INTERNAL TRANSMISSION OF PARASITIC MICROÖRGANISMS

### *General discussion*

The internal forms of disease transmission, in which the parasitic microörganisms either (1) go passively through the insect body or (2) persist and multiply within the body, present greater difficulties for study and demonstration. Furthermore the same organisms that pass through the insect's body are in a large number of instances also carried externally, showing that here, as in all phases of biological science, no hard and fast lines can be drawn. Indeed it is not only possible but probable that in many diseases where only external carriage has hitherto been proved, later researches may demonstrate that one form or the other of internal transmission also takes place.

The simplest method of internal transmission is that in which the organisms merely pass through the body of the insect in the ordinary course of food materials. There are two ways in which microörganisms may leave the body under these circumstances. Many insects are provided with a crop which serves as a storage sac, and such insects commonly regurgitate a part of the contents in order to moisten their food or sometimes as vomit after overfeeding. Ants regurgitate their food in order to feed their young. There are also certain insects that exude liquids from other parts of the body, as in the aphids where honey dew is excreted from nectar tubes. No one so far has demonstrated the liquid excreted from these nectar tubes of aphids to contain microörganisms taken up by the insect, but it is not impossible, and is a question that should be investigated. The ordinary course, however, is for ingested materials to pass through the alimentary tract and out at the anus.

In human and animal diseases there is an abundance of evidence that disease-producing microorganisms can be taken up by an insect and regurgitated in an infective state. The house fly vomit spots have been repeatedly proved to contain infective germs even for a considerable time after ingestion.

Some remarkable facts have come to light in the study of microörganisms taken up by roaches, ants and flies. Cao (26, 1898; 27–28, 1906), an Italian investigator, has perhaps gone as far into the problem as any other one investigator, although every step has been confirmed independently by others. Working with larvae and adults of *Musca domestica* Linn., *Calliphora vomitoria* Linn., *Lucilia caesar* Linn., and *Sarcophaga carnaria* Linn., he proved that the larvae of these four species of flies when breeding in flesh may take up the germs of animal diseases as well as non-pathogenic forms; that some of these germs may pass unaltered through the alimentary tract; that some of them may remain for a long period in the intestinal canal and in a few instances may even multiply therein. Some disease germs may be taken up by the larva, persist through its metamorphosis and for days thereafter, and may be carried by the adult and deposited with its feces on food or dejecta. These bacteria were also isolated from the glutinous envelope surrounding the eggs when deposited, thus contaminating the substance on which the newly hatched larvae were later to feed.

Some bacteria have been shown to be capable of sporification as well as multiplication within the insect, and certain parasitic microörganisms have been found almost normally present in the insect body.

### *Mechanical internal transmission*

In the mechanical internal type of insect transmission the infective principle remains viable after passage through the alimentary tract but undergoes no appreciable multiplication or development within the insect body. Only a passive relation exists here between insect and microörganism.

*Bacterial diseases.* *Bacillus pestis* Kit., the cause of bubonic plague, has been shown by Yersin (173, 1894) and Nuttall (99, 1897) to be capable of remaining in the intestines of flies in a virulent condition for at least forty-eight hours. Infection is usually brought about, however, through the rat flea (*Xenopsylla cheopis* Roths.), which transmits the disease from rat to rat and from rat to man. The bacilli have been found in the intestines in some cases for at least three weeks after ingestion, but it has not been demonstrated that they necessarily multiply or undergo any transformation within the insect. The bacilli are inoculated directly by the flea bite or by scratching portions of the bodies or feces of infected fleas into abrasions in the skin.

*Fungous diseases.* Among fungous diseases, the spores of at least seven parasitic species have been shown capable of passing through the intestinal tract in a viable condition. The fact that flies are attracted to the stinkhorn fungus (*Phallus impudicus* Linn.) has long been known to botanists. T. W. Fulton (48, 1889) in his paper on the spore dispersion of Phalloids by insects, even found a reference to the attraction of flies for the sporiferous slime of this fungus in some latin verses by the old botanist, Hadrianus Junius, who died in 1575. He refers (p. 211) to other such reports by Schäffer (1760), Ventenat (1798), Greville (1826), Berkeley (1857), von Schlechtendal (1862), and Cooke (1875). The first notice of a probable adaptation to insect distribution was given by R. Standen (141) in 1879, and a fuller account (49) the following year by W. R. Gerard. All these notices, however, were more in the way of inference than of systematic investigation. Fulton attacked the problem from a scientific standpoint and demonstrated microscopically that the spores not only cling to the appendages of blow flies but pass through the body apparently unharmed. Then he sowed the sporiferous feces of flies in sterilized soil and obtained typical mycelium of the fungus, whereas in the controls none developed. He considered it probable that in the Phalloids we have a group which has become modified for dissemination by insects, and especially by those insects which habitually frequent putrid substances and would therefore deposit the spores in the most suitable nidus for their growth. It seemed probable to him also that the odor of putrescent matter in general has been developed in relation to insect visits, for the advantage to the bacteria is obvious. He suggests further that flies may not only disperse the bacteria of putrefaction but also those causing diseases such as anthrax.

Istvánffi (63, 1903) speaks of the well known spore distribution of the carrion fungi by insects, and refers to one of the stink-horn fungi, which is reported as causing a root disease of grapes in Hungary..

Cobb (33, 1906; 34, 1909) reported extensive studies of sugar-cane root rot caused by the stink-horn fungus, *Ithyphallus coralloides* Cobb, and related fungi. He proves conclusively that the spores are not only carried externally but that passage through the intestines of insects, particularly species of Sarcophagid and Muscid flies, in no way reduces their viability. The fresh fructification of Ithyphallus has a peculiar, penetrating odor that attracts insects. Flies, beetles, and ants are shown to be very fond of the sticky, dark green spore mass at the apex, and indeed flies sometimes can scarcely be driven away. There seems to be a distinct adaptation benefitting both insects and fungus. Ants take the spores to their underground habitations, where it is fair to suppose that the spores find conditions of moisture and coolness favorable to germination. On the feet of flies spores would become widely disseminated, but by far the larger portion are swallowed and may be found in their stomachs and intestines after they have visited the sporophores. The Ithyphallus is to all intents and purposes a subterranean species, coming to the surface only a few hours for the purpose of advertising its spore product to the insect world. Its wide distribution by this means through the fields places it ever in readiness to take advantage of vulnerable points of attack in the roots of sugar cane.

Worthington Smith in 1884 (135, p. 339) and de Bary in 1887 (14, p. 227–228) noted that the glittering saccharine liquid exuded by *ergot* (caused by *Claviceps purpurea* Tulasne) is eagerly sought after by flies, and they suggest that insects aid in the propagation of this disease. Stäger (138, 1903; 139, 1910) reports a large number of insect species observed by him to visit ergot-infected grasses, and demonstrates that ergot spores are carried on their bodies. Mercier (89, 1911), however, first proved experimentally that flies (*Sciara thomae* L.) not only carry viable ergot spores on the body but also in the alimentary tract, and that they deposit these viable spores in the feces after visiting ergot on rye grass (*Lolium perenne* L.). Mercier captured other insects on diseased rye grass and showed that *Dolerus pratensis* L., *Sapromyza* sp., and *Syrphus decorus* Meig. are also capable of carrying ergot conidia in these two ways, thus completing the experimental work begun by Stäger and showing the exact method of transmission.

According to Wolf (172, 1916), the spores of *Cercospora personata* (B. and C.) Ellis, cause of *peanut leafspot*, are borne by the wind and probably also by insects such as grasshoppers. Out of a total of 75 individual insects collected in 5 different counties of Alabana, this fungus was found externally and in the feces of 54, including 4 different insect orders. Grasshoppers, katydids, and roasting-ear worms (*Heliothis obsoleta* Fab.) were among those giving positive results. The germinating power is not destroyed by passage through the alimentary tract of grasshoppers, and damage by this disease appears to be correlated with the prevalence of this insect in the fields. Grasshoppers are wide fliers and so probably distribute the spores for considerable distances.

Parrott and Fulton (102, 1914) suggested that the snowy tree cricket (*Oecanthus niveus* De Geer) is chiefly an orchard pest on account of the occurrence of cankers caused by *Leptosphaeria coniothyrium* (Fckl.) Sacc. around their oviposition wounds in the bark of apple trees. Later (1916) Gloyer and Fulton (50) in reporting their experimental studies with this disease, show experimentally that not only do tree crickets bear a wound relation to this disease, but that they actively carry the spores of the causal fungus in a viable condition both externally and in the digestive tract and feces. The spores were carried in this manner from diseased to healthy raspberries, and from raspberries to apple trees with resulting infections. Typical cankers in apple branches were also obtained by artificial inoculation with feces of spore-fed tree crickets. It is clearly demonstrated that these insects carry the spores internally and externally and that they also inoculate them into the host. Clinton (32, 1907), in speaking of the *wilt and fruit rot of raspberry* caused by the same fungus, states that in the fruit rot phase, infection undoubtedly takes place through the flowers and very young fruit. He suggests that the fungous spores are probably carried to the blossoms by bees and other insects that have previously crawled over stems coated with the spores, but no experimental evidence is given.

The greenhouse tests of Gravatt and Marshall (53, 1917) show that the spores of the *white pine blister rust* fungus (*Cronartium ribicola* Fischer) are carried externally and in the feces of small arthropods and gasteropods and that they may remain on the body for at least a week. Germination after passage through the insect was in most cases greatly reduced but positive results were obtained from the feces of slugs, sow bugs, cockroaches, weevils and other small animals. Inoculations with the first and second pellets from a weevil gave infection on a western species of Ribes. The following year (1918) Gravatt and Posey (54) proved that gypsy moth (*Porthetria dispar* L.) larvae feed on the Peridermium stage of this fungus and carry thousands of spores on and within their bodies. As Collins has shown that these larvae may be blown as far as 20 miles, their potentiality as disseminators is obvious. That these larvae do actually transmit the disease to Ribes by the uredospores was experimentally demonstrated. Germination after passage through the insect was, however, low. Snell (136, 1919) found Cronartium spores on 40 different species of insects collected on or near white pine cankers, and quite regularly on caterpillars, stink bugs, leafhoppers, rosebugs, etc., taken on infected Ribes. The disease was experimentally induced on Ribes bushes by caging with measuring worms (Geometridae), rose beetles, larvae of a sawfly, a large spiny caterpillar, large ants, and stink bugs, showing that practically all insects that visit diseased and healthy plants may become carriers. The part played by insects confined to the pine host he says, is very limited, but their rôle in the spread of the uredo stage from bush to bush (Ribes) may be, and probably is, an important one.

According to the field observations of Martin (86, 1918) the early blight on tomatoes, caused by *Alternaria solani* (E. & M.) Jones and Grout, is distributed by flea beetles (*Epitrix cucumeris* Harris), as suggested by the fact that beetle punctures are in the centers of early blight spots. Examination of larvae and adults of the Colorado potato beetle (*Leptinotarsa* *decemlineata* Say) and the tomato worm (*Protoparce carolina* L.) revealed spores of *Alternaria solani* and of the leaf blight fungus, *Septoria lycopersici* Speg. on their bodies. Excreta from these and other insects showed the presence of these spores also, and in many cases they were seen to be germinating. It was concluded by Martin that insects are important in the dissemination of these two diseases, but no direct transmission experiments are recorded.

### *Biological internal transmission*

In the biological type of transmission the infective principle finds within the insect body conditions favorable to its development and multiplication. In this way the duration of the period of infectivity becomes greatly lengthened, and likewise the difficulties of observation and demonstration are much increased. Many of the protozoan and so-called filterable virus diseases and a few bacterial and fungous diseases have been shown capable of this prolonged existence and multiplication within the insect. It is relative to this type of insect transmission in both plant and animal diseases that some of the most careful and extended investigations have been carried out.

*Bacterial diseases.* *Bacillus typhosus* Zopf., *B. pestis* Kit., and *Aplanobacter anthracis* (Cohn) Erw. Sm. are sometimes capable of prolonged existence in the intestines of insects, and in some cases at least, *B. typhosus* appears to be capable of multiplication also. Faichnie (40–41, 1909) proved that flies could carry the typhoid bacillus in the intestines for sixteen days. Ledingham (76, 1911) found that the usual intestinal flora of house flies (*Musca domestica* Linn.) generally prevented successful survival of the bacillus through metamorphosis, but in some cases he was able to isolate the organism from the intestines of adult house flies which had been fed on infected material during the larval stage. (It seems evident in the case of these occasional "typhoid carriers," where infection is borne over long periods of time, that an intimate relation must exist allowing multiplication of the bacteria within the insect body.)

Among bacterial diseases of plants an insect relationship somewhat similar to the above given examples is found in *bacterial wilt of cucurbits* caused by *Bacillus tracheiphilus* Erw. Sm. It was stated by Erwin F. Smith (122–123) in 1893 that the spread of this disease is associated with the puncture or eating of the plants by insects, and in 1895 (124) (cf. also 129, v. 1, p. 92, 1905) he experimentally carried over the infection to healthy plants with bacillus-besprinkled striped cucumber beetles (*Diabrotica vittata* Fabr.) and squash bugs (*Anasa tristis* de Geer). In 1897 (127, p. 123) he asserted that in all probability the spread of this disease is almost wholly if not exclusively by insects. In his later (129,

v. 2, p. 209–298, 1911) exhaustive treatise on this disease it is repeatedly demonstrated that the striped and the 12-spotted cucumber beetles (*Diabrotica vittata*, and *D. duodecimpunctata* Oliv.) carry and inoculate the bacillus into the hosts. Experiments with squash bugs (*Anasa tristis*) fed on wilting plants under natural conditions gave negative or doubtful results He suggests from circumstantial evidence that possibly cucumber beetles are not only agents of summer dissemination but also of winter transmission. Field cage experiments by Rand (106, 1915) in a locality free from wild cucurbits gave no infection from soil, or by flea beetles (*Epitrix cucumeris* Harr.) and aphids (*Aphis gossypii* Glov.); but gave positive and widespread infection by striped cucumber beetles in the experimental fields at large, but in cages only where these insects had been introduced. After caging with the first striped beetles to appear in spring healthy young cucumber plants gave wilt infection, starting from their gnawings. These beetles had been collected some time before wilt appeared in this locality. Later experiments by Rand and Enlows (107, 1916) abundantly confirmed the previous exhaustive studies of Smith in incriminating striped and 12-spotted cucumber beetles as direct summer carriers. All tests with aphids, flea beetles, squash lady-birds (*Epilachna borealis* Fabr.), and with seed or stomatal infection gave negative results. In these and later studies (108, 1920) a direct mathematical relation was shown to exist between prevalence of striped beetles and the spread of the wilt disease. It was also shown that insects and nematodes do not carry infection from soil to host plant, and that root infections do not occur under field conditions. Complete locality records proved that the first spring cases start on occasional plants from gnawings of cucumber beetles which in these cases must have recently emerged from hibernation. All subsequent cases developed from similar injuries, and the disease tended to spread in ever widening circles around the initial cases. It was furthermore proved that the striped beetles are sometimes capable of infecting at least 3 to 4 healthy plants successively fed upon after one initial wilt feeding, and that after one such feeding they may, in the active state, retain their infecting power for at least twenty-three days. Occasionally wilt-fed beetles retained their infective power after five or six weeks in cold storage, i.e., as long as they were able to keep them alive. Dissection and inoculation experiments from intestinal contents proved that a small percentage of both striped and 12-spotted beetles may harbor the bacillus in the alimentary tract. In a series of isolations of bacteria from the striped beetle the intestinal flora of one only out of the 10 individuals tested was found favorable to the wilt organism. In the other 9, *Bacillus tracheiphilus* was not present. Greenhouse inoculations of 10 plants from the viscera of these 10 beetles also gave 1 case of wilt. Recent studies by Rand and Cash (109, 1920) have confirmed these results Furthermore, they also experimentally proved that infections take place directly from beetle droppings brought into contact with freshly pierced leaf surfaces. In these cases oral contamination was prevented. In their experiments also inoculations of intestinal contents have given wilt from the first beetles to appear in the spring, and fifteen to eighteen days before field cases had appeared in the vicinity. Also *Bacillus tracheiphilus* has been isolated from the viscera of a certain percentage of wilt-fed beetles and later successfully tested on cucumber plants. From these facts it appears legitimate to conclude that this bacillus is capable of continued life and multiplication within the alimentary canal of a certain small portion of wilt-fed beetles which may be designated as "wilt carriers." There is also strong circumstantial evidence that the organism survives the winter in this manner.

Relative to the *olive-knot* organism (*Bacterium savastanoi* Erw. Sm.), Erwin Smith stated in 1908 (130, p. 42) that old tubercles are eaten by various insects, and he considered it possible that some insect carrier may play a part in the distribution of the disease. The following year Petri (104, 1909) reported his exhaustive studies on this phase of the problem and showed that a definite symbiotic relation exists between *Bacterium savastanoi* and the olive fly, *Dacus oleae* Rossi. He found bacteria constantly localized in the fourth gastric pouch of the larval midgut. The presence of these bacteria was demonstrated from the moment when the larvae emerged from the egg up to a point immediately preceding the pupal stage. The position of the bacteria in this blind sac was such that during metamorphosis they were not expelled as a whole, but as they increased enormously in numbers some of them were from time to time passed out of the anus in the ordinary course of ingested food. Neither bacteria nor other microorganisms were found in the salivary glands or the malpighian tubules of the larvae. Immediately preceding the pupal stage, the gastric pouch and all the rest of the digestive tract were almost completely emptied. A few bacteria, however, still remained near the lower aperture of the cardiac valve and they became passively transported into the esophagus, where, between the folds of the cuticle, they remained localized throughout almost the whole period of development. With the full development of the adult fly the course of the bacteria was lost for a short period of time, varying according to the season of the year. However, the bacteria appeared irregularly a little later in a glandular sac above the pharynx. Petri did not succeed in cultivating *Bacterium savastanoi* from the pharyngial sack, but yet was able to produce the tubercles in olive by inoculating its contents. After the emergence of the adult these bacteria again suddenly developed at the expense of the abundant secretions of the epithelial cells lining this gland, and from this point they rapidy diffused into the midgut where they were later found in ovoidal groups. In the female the bacteria were also localized in numerous small glands near the anal opening which is situated on the ventral side of the ovipositor. The development of the bacteria appears to be independent of the amount of food taken in by the insect, but to bear a direct relation to its functional activity. Thus, in the female emerging in winter not only are the eggs very slow in forming but the bacteria in the pharyngeal gland are also slow in development. The transmission of the bacteria from the female to the egg occurs through the ejection of a small quantity of bacteria from the anus at the same time that the egg is deposited in the olive branch. Thus are these microorganisms distributed over the surface of the egg at the moment when it passes through the cloacal canal of the ovipositor. During the development of the embryo within the egg the bacteria multiply actively around the micropile in the gelatinous envelope, and later penetrate the egg by passing through the chorion and through the numerous air canals surrounding the micropile. The bacterial content of the blind gastric pouch of the larva is not a pure culture of one species of bacteria alone. However, *Bacterium savastanoi* is present on the egg and in the micropile, in the gastric pouch of the larva, in the midgut, and in the pharyngeal glands of the adult, and in the anal glands of the female. These facts were shown by direct inoculations into olive branches followed by development of typical olive-knot tubercles and isolation of the causal organism from them. In a few cases Petri also succeeded in isolating the olive-tubercle organism directly from the gastric pouch of the larva and from parts of the digestive tract of the adult, but it occurs here sparingly in comparison with saprophytes. The bacteria subsist on the glandular secretions of this insect and on the death of the host all further development of the bacteria ceases. Petri concludes that *Bacterium savastanoi*, cause of olive-knot, is the normal symbiont in the digestive tract of *Dacus oleae*, and that the yellow organism (*Ascobacterium luteum*) often associated with it in great abundance, is a follower. Horne, Parker, and Daines, (60–61, 1912), working in California, found no biting or piercing insect concerned in the dissemination of the organism there, and concluded that the distribution is adequately accounted for by the spattering of rain and the accidental tracking of birds, insects, etc.

Tonelli (155, 1913) working with a *bacteriosis of oleander*, concludes that the scale insects, *Aspidiotus hederae* Vall. (*A. nerii* Bouché) and Lecanium sp., and the mite, *Tetranychus telarius* Linn., with certainty carry infection on their mouth parts and directly inoculate it into this host. He also considers it probable that a species of Diptera frequenting the flowers may carry infection to the stigmas. Both Smith and Petri agree, however, that *Bacterium savastanoi* when inoculated into the oleander does not produce tumors, and therefore cannot be the cause of the oleander tubercle as is claimed by Tonelli

Johnston (67, 1912) states that the field evidence all points in the direction of insects as instrumental in disseminating *coconut bud-rot* caused by *Bacillus coli.* He isolated the bacillus from the intestines of earwigs and from the droppings of turkey buzzards; and considers it probable from his studies that insects are carriers of infection. (Forms of *Bacillus coli* have from time to time been isolated from the alimentary tracts of insects as apparently normal intestinal inhabitants. The insect relation to this disease presents an interesting problem.)

*Fungous diseases.* Berlese (15, 1897) has shown how some insects, particularly ants and flies, contribute to the diffusion, preservation and multiplication of certain yeasts and molds. He demonstrated that ants may carry yeasts and molds up from infected soil and successfully inoculate grapes on the vines above. Flies fed upon yeasts gave these yeasts again in cultures from the feces, while flies fed upon killed yeast cultures yielded negative results. Berlese also demonstrated that yeasts multiply within the bodies of flies, and he found the sugary solution in the crop an ideal situation for their development. As a result of these studies he considered that during the cold season some yeasts are chiefly preserved and perhaps increased within the bodies of insects.

Schneider-Orelli, in 1913 (116), found a symbiotic relation existing between the beetle, *Xyleborus pyri* Peck. (formerly *X. dispar* Fab.), and the fungus, *Monilia candida* Hartig. He demonstrated by cultural methods that the female beetle always carries in the crop a store of living fungous cells which are not used up in her metabolism. Even when kept without food for two and a half months the living fungus was still found to be present in the crop. Shortly after the boring by the female into the wood of orchard trees the passage becomes covered with a fleecy white growth of the fungus which develops inward with the extension of the passage. Under normal conditions this nutrient fungus is present in practically pure culture, but with the growth of the larvae in the passage the fungous layer disappears and at the time of hatching only the black walls of the passage are left. Schneider-Orelli concludes from his experiments that the fungus itself is not an important factor in injuring the tree, as artificial borings alone resulted in the death of young trees in the same manner as from the insect borings. Inoculations with the fungus alone caused little or no browning of the adjacent wood in healthy, growing trees.

*Protozoan diseases.* The relation of the various forms of human malaria to Anopheles mosquitoes is too well known to need discussion. Many other instances of biological transmission of protozoan diseases of man and animals by insects are on record. Such, for example, are the trypanosome diseases transmitted by the tsetse fly in Africa, viz., the *sleeping sickness* of man, caused by *Trypanosoma gambiense* Dutton, and *nagana* of ruminants, caused by T. *brucii* Plimm. & Bradf.

*Spiroschaudinnia duttoni* Novy & Knapp, the cause of African *relapsing fever*, is taken up by the horse tick (*Ornithodorus moubata* Murray), which lives in the mud floors of huts or in the soil where caravans habitually camp. The spirochaetes have been demonstrated in the ovaries and eggs of female ticks and have been experimentally carried to the third generation, showing a true hereditary transmission. Furthermore, it is said that the bite of individual ticks may still be infective one and a half years after an initial feeding on an infected host.

There is at least one instance of a protozoan plant disease in which the parasitic organism passes one phase of its life history in an insect. It was demonstrated by Lafont (74, 1910) that *Leptomonas davidi* Lafont causes a "*flagellosis*" in the latex of several species of Euphorbia. This disease is transmitted by the bug, *Nysius euphorbiae* Horvath., which is the permanent alternate host. The genus Leptomonas is composed of protozoa which are almost exclusively insect in their origin, although some of them have been shown to be pathogenic to vertebrates.

*Filterable contagium diseases.*[1] Among both plants and animals the so-called filterable virus diseases are of fairly common occurrence. Some of the diseases formerly classed as "filterable virus" diseases have since been demonstrated to be due to parasitic microörganisms. In all this class of infections the method of contraction and the sequence of signs or symptoms give a "clinical picture," so to speak, of a parasitic disease. The only difference is that in the "filterable virus" diseases the causal contagium has thus far eluded our microscopical and cultural methods.

Among animal diseases of this type *yellow fever* is a well known example. This dread scourge of the tropics is borne by mosquitoes, and the ultimate contagium will pass through the pores of a Berkefeld filter. The contagium of yellow fever circulates in the blood stream of a patient for at least three days after the initial chill. To become infected a female Aedes mosquito (*Aedes calopus* Meig.) must bite a fever patient during this first three days, whereupon a latent period of twelve days must then elapse before the infected mosquito can transmit the disease. Injection of blood from a fever patient during the first three days will induce the disease in a non-immune person; otherwise the only known method of transmission is through the bite of this mosquito. (Noguchi, through a careful and extended investigation, has recently shown yellow fever to be due to *Leptospira icteroides* Noguchi. This serves only to emphasize the statements made in the preceding paragraph.)

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[1] The term "contagium" has been suggested by Dr. H. M. Quanjer as synonymous with any *infective principle*, whether of known or unknown origin. Its use in the place of "virus" with reference to the so-called "filterable virus" disease does away with objectionable connotations, and leaves nothing to be taken back.

In the plant kingdom those diseases classed as "infectious mosaics and chloroses" easily fall under the heading of filterable contagia. The first of this type of plant diseases demonstrated to be transmitted by insects is "*curly leaf*" or "*curly-top*" of *sugar beets.* On circumstantial evidence E. D. Ball in 1906 (8) connected this disease with the beet leafhopper (*Eutettix tenella* Baker) as a cause. He states that the injury does not appear to be due to the amount of sap removed, but that the punctures of this insect seem to cause a thickening of the leaf veins and an unhealthy condition of the plant. He refers also to other species of this group of leafhoppers as causing somewhat similar disturbances in other plants, particularly in the common pigweed. In 1909 (9) he went over the results of his earlier work somewhat more in detail, stating that the greatest abundance of curly-leaf followed the greatest prevalence of beet leafhoppers, and that the signs of disease induced by this leafhopper are specific. In sugar beets the abnormal condition apparently spreads until the whole plant becomes involved, even though the leafhoppers have long since disappeared. The disease was found also to persist the following year in the root. Spisar (137, 1910) refers to the work of Ball but gives no new evidence. Shaw. (121, July, 1910) gives the first definite experimental proof that curly-top is directly transmitted by the beet leafhopper. In 1908, at Townsend's suggestion, Shaw sent beet leafhoppers to Washington, D. C. where cage experiments demonstrated the disease to be transmitted by this insect. These cage experiments were repeated and amplified many times by Shaw in the field. and always with the result that the disease was transmitted by beet leafhoppers but not by aphids, thrips, or red spiders. The nymphs appeared more virulent than the adult leafhoppers. Curly-top beets were never found to recover but seed from such beets gave healthy progeny. It was not found possible to communicate the disease from an affected to a healthy plant except through the intermediary of a leafhopper. No outbreaks of curly-top are known outside of the native habitat of *Eutettix tenella,* and no other insect or species of leafhopper is known to induce this disease. Shaw says that an active, persistent agent is introduced by this insect, which sets up disturbances through the whole plant. R. E. Smith and Boncquet (132–133, 1915) repeated and confirmed the work of Ball and of Shaw. They further showed that one leafhopper confined upon one leaf of a sugar beet plant for five minutes was sufficient to induce the disease. They also carried over the disease by grafting a portion of a curly-top root into a healthy root. Townsend (156) in 1915 asserted that so far as determined, *Eutettix tenella* is the only carrier of curly-top, but that there are instances where leafhopper infestation does not produce the disease even though external conditions seem favorable. He then asks this question, "Are the beets sometimes resistant or is *Eutettix tenella* sometimes free from the exciting agent?" R. E. Smith and Boncquet (133, p. 340, 1915) conclude from their experiments that no more than three hours feeding is necessary for the leafhopper to obtain the pathogenic factor, but that at least 24 and not more than 48 hours must elapse after feeding before the insect can induce the disease. These facts suggested to these investigators that curly-top is not carried mechanically but that some development takes place within the body of the insect during the first few hours after feeding on a diseased plant. At the same time Boncquet and Hartung (16, 1915) report convincing experiments with *Eutettix tenella* obtained from wild plants (species of Artemisia and Atriplex) in the Tulare Lake region of California. These leafhoppers, when caged with healthy sugar beet plants, did not induce curly-top, while in control plants caged with local leafhoppers from diseased beets the disease was readily induced. After three weeks 60 of these "wild" leafhoppers were caged with curly-top beets and subsequently transferred to 60 healthy plants. Out of these 60 plants 50 developed typical curly-top while the remaining 10 were doubtful cases. This clearly demonstrates that the leafhopper is not in itself infective, but only after feeding on diseased plants. Ball, in 1917 (10) reported in detail upon his own work and that of other investigators relative to curly-top and its transmission. Boncquet and Stahl in 1917 (17) showed by experiment that beet leafhoppers previously fed upon curly-top beets could transmit the disease to mallow (*Malva parviflora* Linn.). Stahl and Carsner (140) in 1918 obtained nymphs non-virulent as to curly-top by collecting them as they emerged from the egg and before feeding. However, after feeding on curly-top beets these same nymphs were able to transmit the disease. Finally Carsner (30), in 1919 has demonstrated by careful and detailed experiments that a number of wild plants are susceptible to the curly-top disease, that virulent leafhoppers retain their ability to infect for a long period after removal from susceptible plants (e g., 58, and 111 days), and that at least some of the leafhoppers are infective when they enter the beet fields in spring. He asserts that there is substantial evidence that the adult leafhoppers which leave the beets in the fall are not those which appear on beets in the spring, but individuals of a new brood developed on wild vegetation. From these facts he considers there is no question as to leafhoppers retaining their virulence long enough in the fall to infect susceptible wild annuals, such as filaree (*Erodium cicutarium* (L.) L'Her.), which in California appears soon after the first fall rains. Some of the leafhoppers developing on these infected wild hosts undoubtedly secure from them the contagium to infect young beet plants in the spring. "Thus, it seems highly probable, the overwintering of the virus is accomplished."

The connection of insects with *mosaic of tobacco* has been carefully worked out by Allard. He first noted (1, 1912) that under controlled conditions in the course of greenhouse experiments with this disease, healthy plants were always obtained when grown in screened cages frequently fumigated to prevent aphid infestation. On the other hand, when colonies of aphids present in the greenhouse were introduced into cages with healthy plants the result was a wholesale occurrence of mosaic. In 1914 (2) he reports the spread of tobacco mosaic in seed beds and fields, which was unquestionably associated with aphid infestation (*Macrosiphum tabaci* Perg.). By direct experiment (4, 1917) he then showed that the peach or spinach aphis (*Myzus persicae* Sulz.) may actively transmit the infective principle but not until after the insect has fed upon diseased plants. Tests with a large green aphis (*Macrosiphum lactucae* Kalt.) from lettuce gave negative results although they readily fed and multiplied upon diseased and healthy tobacco plants. Similarly pelargonium plant lice (*Macrosiphum pelargonii* Kalt.) failed to carry the infective principle from diseased to healthy plants. This species, however, would not feed upon tobacco and hence could not be maintained for long under the experimental conditions. Preliminary tests with white fly (*Aleyrodes vaporarium* Westw.) and with red spider (*Tetranychus telarius* Linn.) gave negative results. Allard finally observes that Preissecker in 1903 and 1905 had noted the association of tobacco mosaic with attacks of plant lice, but that it does not appear from his writings that he looked upon these insects as actual carriers of the infective principle of the disease.

Allard (1, 1914; 5, 1918) considers it very probable from his observations and experiments on mosaic of pokeweed (*Phytolacca decandra* Linn.) that this disease is also insect transmitted.

Again, (3, 1916) from circumstantial evidence together with his experimental proofs in the case of mosaic in tobacco, Allard considers it probable that *tomato mosaic* is also transmitted by aphids.

Taubenhaus (153, 1914) reports experiments with *mosaic of sweet pea* in which aphids collected both from diseased plants and from the field at large gave mosaic to healthy plants. Sweet pea plants inoculated by needle prick from diseased plants developed mosaic while those inoculated from healthy plants remained healthy.

By careful experiments Doolittle (37), and Jagger (64), working independently (1916), showed that aphids (*Aphis gossypii* Glover, used by Doolittle) collected from plants with *cucumber mosaic* transmit the disease to healthy plants, but that aphids fed upon healthy plants do not carry infection. Transmission by striped cucumber beetles was later (1918) demonstrated by Doolittle and Gilbert (37a).

Loren B. Smith (131, Feb., 1918) reported on experimental evidence that *spinach blight* is a specific communicable disease which is transmitted to healthy plants by insects. Shortly afterward McClintock and L. B. Smith (80, July, 1918) together published a detailed account of their extensive studies of this spinach disease. Healthy plants were successfully inoculated by needle pricks with the contagium from diseased plants and with the crushed juice of aphids fed upon diseased plants. The potato aphid (*Macrosiphum solanifolii* Ashmead) and the spinach aphid (*Rhopalosiphum persicae* Sulzer), free from infection at first, were demonstrated to transfer the blight to healthy spinach after feeding upon diseased plants. Control plants invariably remained healthy. Later these two species were obtained from 4 different states where spinach blight did not occur and they failed to induce the disease on healthy plants until after they had fed on blighted spinach. The same two species collected locally and tested at the same time produced the disease. It was experimentally shown that a small percentage of the offspring of aphids fed upon blighted plants, though born and reared on non-susceptible species of plants, may prove infectious to spinach. The juices of these non-susceptible plants invariably gave negative results upon inoculation into spinach. It is evident that the infective principle is capable of transmission from parent to offspring. Even after molting a "virus-bearing" aphid remains infective, showing that the contagium is carried internally. Furthermore, successful inoculations were obtained from 4 generations representing 3 strains and 2 species of aphids, after one feeding of the parents on diseased spinach. However, the incubation period of the disease is longer with the later generations. It was also demonstrated that not *all* individual aphids fed upon diseased spinach are "virus carriers." It was further proved that aphids (*Macrosiphum solanifolii*), after feeding on wild mustard, a non-susceptible plant, were still infective although neither they, nor their parents for a long time, had fed on spinach. Inoculations with the juice of mustard plants gave a negative result. These investigators demonstrated that the contagium may be carried from spring to fall by a direct line of aphids, and that non-virus bearing aphids must remain on diseased plants 5 minutes to 14 hours to become carriers. The bean aphid (*Aphis rumicis* Linn.) and tarnished plant bug (*Lygus pratensis* Linn.) gave infections after feeding on blighted spinach. Negative results were obtained, however, with the adult of the southern corn rootworm (*Diabrotica duodecimpunctata* Olivier), the green plant bug (*Nezara hilaris* Say.), *Thrips tabaci* Lind., *Smynthurus hortensis* Fitch, and *S. quadrimaculatus* Ryder. McClintock and Smith compare their findings with the results of animal disease investigations, and conclude that spinach blight, together with the "filterable virus" diseases in general, are due to ultra-microscopic organisms.

In greenhouse experiments it has recently been shown by Schultz et al (117, 1919) that potato mosaic may be transmitted to healthy plants by aphids after feeding on plants affected by the disease. Untreated plants and those treated with non-virulent aphids remained healthy. It should be added that all three groups of plants came from the same tubers, thus precluding the possibility of "seed" transmission in the positive cases recorded.

Ball has recently (11, 1919) reported a series of observations and experiments upon a potato disease which may be tentatively included here. He has clearly demonstrated its dependence upon the potato leafhopper (*Empoasca mali* LeB.), and given the name "hopperburn" to the disease. This species of leafhopper, and this species alone, is shown to cause similar signs on several hosts, while the effects of other leafhopper species on these same hosts is entirely different. Whether or not hopperburn will prove to be a specific transmissible disease like curly-top of beets remains to be demonstrated, but in any case its relation to the potato leafhopper appears to be a specific one. In a later report of the same year (12, 1919) Ball reports the recovery of a caged potato plant after the removal of the leafhoppers, while during the same time the disease continued to spread in the field at large. Furthermore, this disease does not appear to spread from one part of the plant to another as in the case of curly-leaf of beets, and it must therefore be quite different in nature.

## CONCLUSION

The literature on plant diseases transmitted by insects is thus seen to be fairly extensive, and the importance of insects as disseminating agents becomes from year to year more apparent. In fact, the investigations of the past three decades have completely revolutionized our view of the rôle of insect transmission in both plant and animal diseases. However, in many cases authentic information is still lacking as to the exact methods of inoculation and transmission, and in some instances too much is left to inference and circumstantial evidence; but enough really good work has been done to show an exact correlation between the findings of plant and animal pathology. Furthermore, it is not to be doubted that future work will fill up many of the existing gaps in our knowledge and will add greatly to the list of insect borne diseases.

In many problems of insect transmission unquestionably the best results will follow a collaboration between pathologist and entomologist. In order to follow such problems to a scientific and logical conclusion a great amount of exceedingly detailed work is necessary, not only with the host plant and its parasitic microörganism, but with the disseminating agent as well. It is necessary to know the habits and the anatomy of the insects concerned. Furthermore it is often imperative to breed uninfected insects to act as agents of transmission and as controls on an experiment. A pathologist can hardly expect to know all the technique of breeding insects any more than an entomologist can expect to know the technique of culturing microorganisms.

In any case of suspected insect transmission the first step is to determine whether the contagium is carried on the insect's body. In some diseases this may be determined microscopically. In other instances, such as the bacterial diseases, it may be necessary to culture the organism. This may be done by washing the body, or separate parts of the body, in a sterile fluid medium and subsequently plating out the organism. In all cases where possible this should eventually be done in order to determine how long the parasite retains its virulence on the body of the insect.

A second series of experiments should determine whether the parasite retains its virulence after passage through the alimentary tract of the insect. Attempts should be made to culture the organism from the intestinal contents and from the feces; and in many cases the host may be inoculated directly from these sources.

In the case of many plant-feeding insects there is a progressive movement over the host, and if the insect is in the habit of defecating at the time of feeding it is likely that the feces will be dropped sooner or later directly on an abrasion which the insect itself has made. Experiments should be devised to determine whether the dropping of contaminated feces on a fresh injury will induce the disease in the host. It is, of course, imperative to preclude the possibility of contamination from the mouth parts or other sources. In some cases insects known to be non-infective may be first used to injure the host. Furthermore, in some diseases sensitive parts of the host may become infected without the intervention of injuries of any sort.

Other internal organs of the insect, such as the salivary glands or the crop must sometimes be tested for the presence of the parasite. In many cases this will involve the devising of special dissecting and culturing methods. In all cases, so far as possible, both cultures and direct inoculations should also be made with the contents of each organ studied.

It is important to determine whether the parasite can be transmitted directly by the bite of the insect, and in the case of positive results to find whether the contamination is merely from the external mouth parts or from the salivary glands or portions of the alimentary tract. Internal oral contamination may sometimes be determined by sterilizing the external portions of the body and mouth parts and allowing the insect to feed upon the host without defecation. In other cases cultural or microscopical methods must be used.

Among the points to be studied, then, are the following: It is necessary to determine on or in what part or parts of the body the contagium is carried: whether the transmission is mechanical or biological; how soon after taking up an infective principle transmission is possible; how long the insect remains infective; whether an infected larva may retain the contagium through its metamorphosis; whether the contagium can be transmitted to the offspring, and if so, for how many generations; whether the offspring can transmit the disease at any stage of its development; whether an infected insect remains infective after a period of feeding on non-susceptible hosts; and whether the contagium winters over in the insect. The mere fact, however, that the contagium of a disease is found in or on the body of an insect should in no case be taken as final proof of an insect relation to transmission. In many diseases an inoculating needle, a piece of wood, or *anything* which happens to come into contact with the diseased tissues will carry upon it some of the contagium. *The final criterion, then, should be the actual transmission of the disease under controlled conditions simulating as nearly as possible those found in nature.*

The task of working out all these varied ramifications of the problem in the case of any specific insect transmitted disease will require a good technique and the utmost care and patience on the part of the pathologist. Much may be learned from animal pathologists and a direct collaboration with entomologists will often eliminate pitfalls and facilitate the attainment of the ultimate goal.

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# THE PHYLLOSTICTA BLIGHT OF SNAPDRAGON[1]

EDWINA M. SMILEY

WITH EIGHT FIGURES IN THE TEXT

While examining benches of snapdragon in greenhouses at Batavia and Corfu, New York, in the spring of 1917, certain plants were found to be infected with a disease the symptoms of which closely resembled those of the anthracnose. On further study, these symptoms proved to be those of the Phyllosticta blight. The same disease was observed in gardens at Ithaca, New York, during the summer of 1917, and again in 1918, on both garden and greenhouse plants. As far as is known the commonly cultivated species of the snapdragon, *Antirrhinum majus* L., which has been known to gardeners for centuries, is the only host affected by this disease. All of the thirty or more varieties of the species which have come under the observation of the writer showed susceptibility to the blight, but no detailed study has been made of their relative susceptibility. It was observed, however, during the summers of 1917 and 1918, that the leaves of the red-stemmed varieties such as the Giant Garnet are severely affected, while those of the green-stemmed varieties like the Yellow (intermediate) and Silver Pink are attacked slightly or not at all. The reverse is true of the stems; lesions on the green stems are larger and more numerous than those on the red stems. These observations were made on plants which were growing in the same garden under the same conditions.

Similar observations were made in the case of artificially inoculated greenhouse plants. Infections on stems of the Giant Garnet, for example,

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[1] Also presented to the Faculty of the Graduate School of Cornell University, May, 1919, as a major thesis in partial fulfillment of the requirements for the degree of Master of Arts.

After the completion of this manuscript and its presentation for publication, a paper on the same subject by E. F. Guba and P. J. Anderson appeared in Phytopathology for August, 1919. Since this work was done quite independently, the writer having no knowledge of the others' work, and the two sets of results being for the most part in agreement, it seemed best to publish this paper without change in the original manuscript.

Acknowledgement. The writer wishes to express her indebtedness for the helpful criticisms and suggestions given by Professors H. H. Whetzel and the late V. B. Stewart, under whose direction the work was conducted, and for the painstaking care of Mr. W. R. Fisher in the making of the photographs.

are secured with difficulty, while on stems of the Silver Pink, Yellow, etc., they are easily obtained. The green leaves of the latter forms are more difficult to infect than the red leaves of the first named.

## THE DISEASE

The Phyllosticta blight was first described by Sydow[2] in 1899 as a spot occurring on the leaves, usually at tips or margins, of *Antirrhinum majus* in Steglitz, Germany. In America it was first recorded in 1900 by Stewart,[3] who gave it the names "stem rot" and "branch blight," preferring the latter as more descriptive. Stewart, however, describes only the

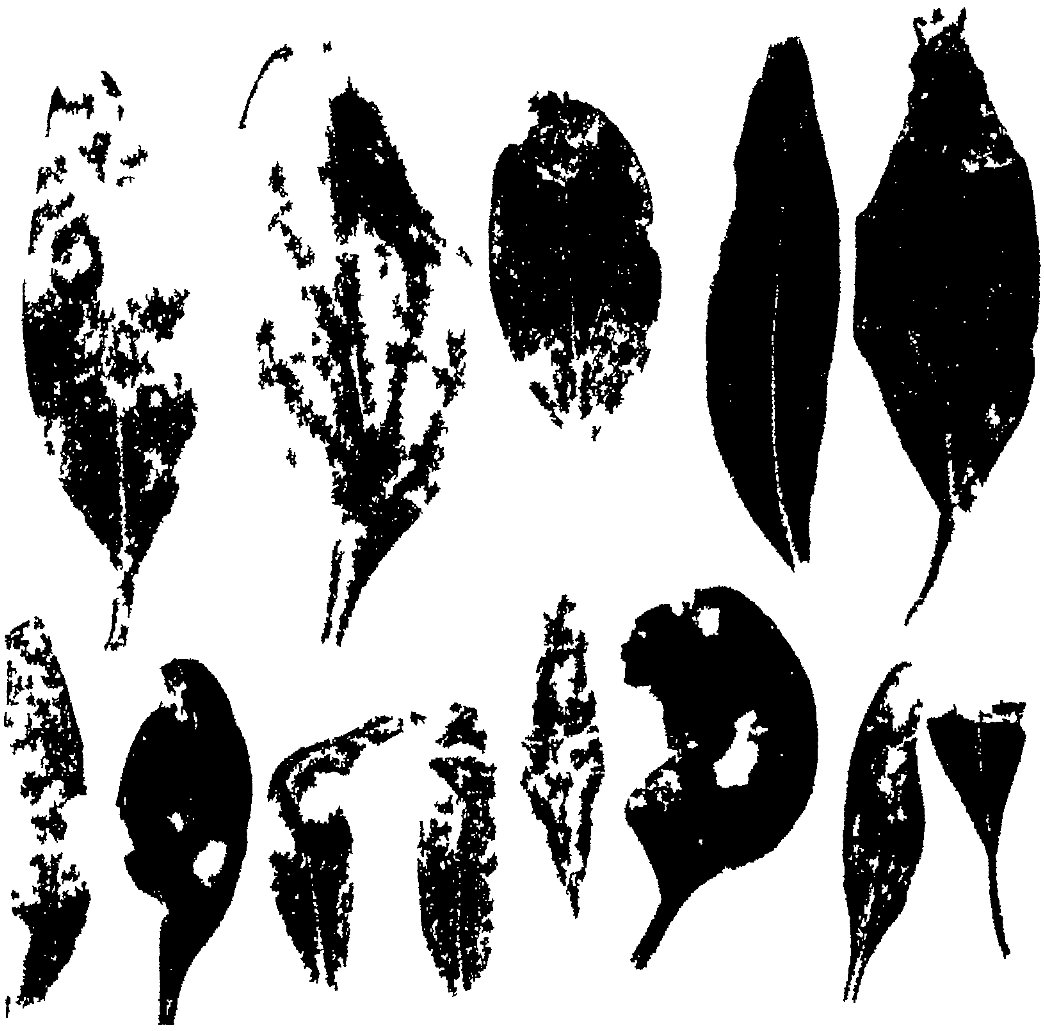

FIG. 1. SNAPDRAGON LEAVES AFFECTED WITH PHYLLOSTICTA BLIGHT
Various types of lesions and their zonate character are shown

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[2] Sydow, P. Beitrage zur Kenntniss der Pilzflora der Mark Brandenburg II. Hedwigia 38: Beiblatt (134). 1899.

[3] Stewart, F. C. An anthracnose and stem rot of the cultivated snapdragon. New York (Geneva) Agr. Exp. Sta. Bul. 179: 109–110. 1900.

browning and dying of the branches, making no allusion to whitish oval spots on the stems and leaves. The disease has since been observed in Indiana and several times in Geneva, New York (F. C. Stewart, letter to writer). It has lately been reported from Massachusetts by Anderson,[4] who suggests the possibility that the diseases described by Sydow and Stewart are identical. Sydow did not mention the occurrence of the disease on the stems and, with the exception of Anderson's suggestion, no report of the relation between the stem blight and leaf spot stages, as far as is known to the writer, has been made in literature. Both forms were found on garden and greenhouse plants observed at Ithaca, New York, during the summer and fall of 1917 and 1918.

In garden plants the chief importance of the disease lies in the unsightly appearance of the spotted leaves and brown, withered shoots. In greenhouses, where the snapdragon is grown on a large scale for bedding plants or for cut flowers, the unsalable condition of the dwarfed young plants or the reduction in blossom yield due to the killing of the branches may result in considerable loss.

## SYMPTOMS

The lesions appear on out-of-door plants in late June or early July. On greenhouse plants they appear in the late fall, when the seedlings or cuttings have reached a height of 6 or 8 inches. Plants of any age are susceptible, but very young seedlings are not often attacked.

*On the leaves.* The effects of the disease on the leaves are various (fig. 1). The first evidence of its presence is the appearance of small dark spots, brownish purple in color on the red leaves, dull greenish brown on the green leaves. These spots most frequently occur at the tip of the leaf or on the margin near the tip. Sometimes, however, lesions may occur in the lamina, in which case they are nearer the base than the tip. The spots are visible on both sides of the leaf and soon become cream-colored and slightly sunken in the center. As the lesions spread, the cream color changes to pale brown, the advancing margin remaining dark. The older lesions are distinctly zonate with fine raised ridges limiting the zones, and the upper surface is thickly dotted, especially toward the center, with small black bodies easily visible to the naked eye. If the tip of the leaf is affected, usually only the one lesion is present, but the margin of this lesion often advances downward into the blade rapidly and irregularly until more than half of the leaf is involved. If the early lesions appear in the lamina, one or several may occur. These spots are elliptical, though the margin may soon become irregular and often several spots coalesce forming a large indefinite discolored area. As the lesions become older the diseased tissue becomes so papery and brittle that it breaks away from the healthy tissue giving the leaf a ragged appearance as though it had been eaten by insects. This peculiar brittleness of the affected tissue causing it to crumble to powder when rubbed between the fingers is one of the constant symptoms of the disease. In the case of the smaller lesions in the lamina, the center sometimes falls away producing a shot hole effect. Severely affected leaves may become completely withered, turn dark brown, and cling to the stems for a long time resembling the leaves of the blighted branches. Several cases of the spreading of the infection from badly diseased leaves through the petiole and into the stem have been observed but this does not appear to be of constant occurrence.

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[4] Anderson, P. J. Index to American species of Phyllosticta. Mycologia 11: 66–79. 1919.

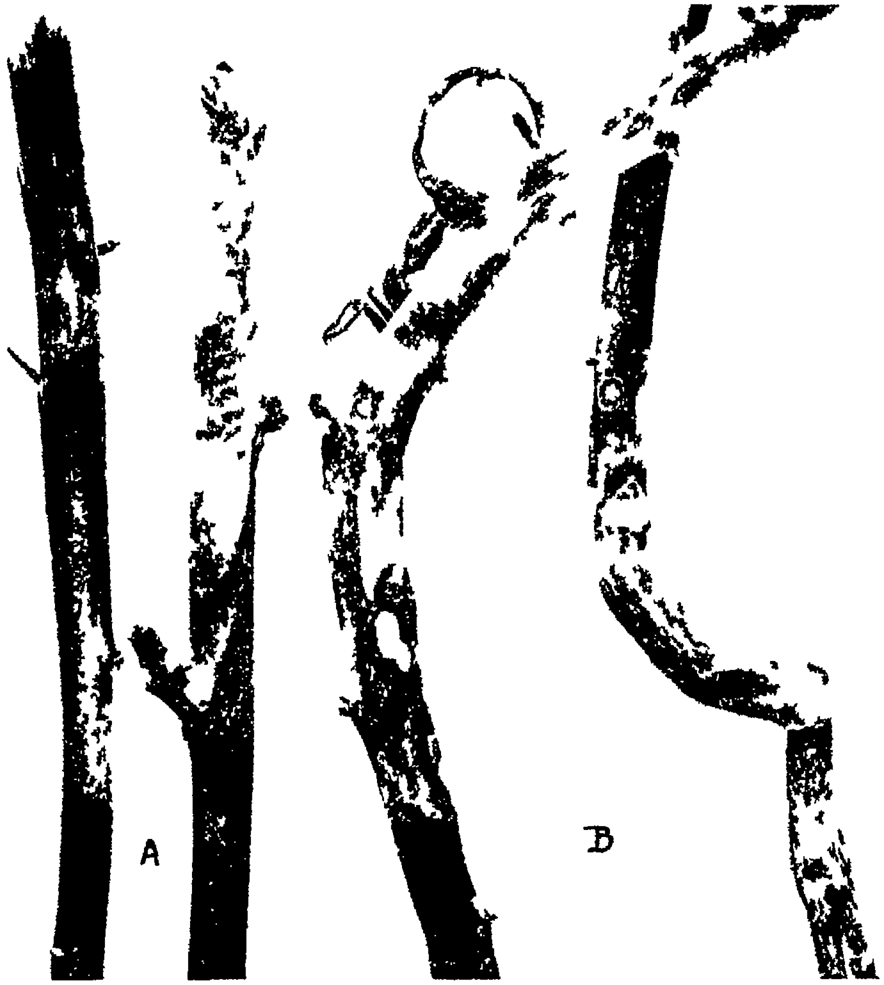

Fig. 2. Snapdragon Stems Affected with Phyllosticta Blight

*A*, Young stems with large brown lesions showing abundant pycnidia. *B*, Old stems showing white spots at base.

*On the stem.* Both old and young stems may be attacked, the lesions occurring at any point and often appearing some time before any leaves are attacked. Stewart, however, says that it is chiefly the succulent shoots which are attacked. Observations of the writer have confirmed this statement for the branch blight form of the disease, that to which Stewart had reference. A water-soaked area suddenly appears on the young shoot several inches back from the tip and spreads rapidly around and lengthwise of the stem. In a few days the affected tissue shows a rather firm dark brown rot, then soon collapses and dries. Sometimes the whole shoot is affected and becomes very brittle, the leaves also turning brown and shriveling. At other times the spread of the water-soaked area is checked for some reason, leaving the terminal portion of the shoot green. In this case the collapsing and drying of the rotted area soon cuts off the water supply to the green tip so that it gradually wilts, often fall-

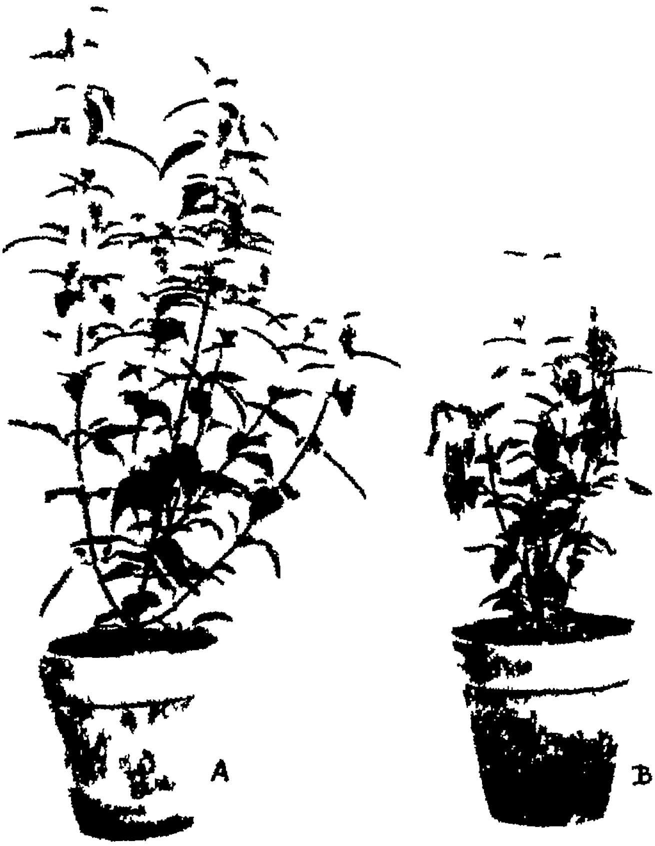

FIG. 3. HEALTHY AND DISEASED SNAPDRAGON PLANTS

A, Healthy plant; B, diseased plant of the same age showing branch blight form of the disease and general dwarfing.

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[*] New York (Geneva) Agr. Exp. Sta. Bul. 179: 109.

ing over at the affected point. These were the symptoms which led Stewart to call the disease "branch blight" and "stem rot" (fig. 3b).

In addition to this blight or rot of the branches a form of the disease occurs in which more definite lesions are produced on the stems. These lesions are first visible as dark green, water-soaked and slightly sunken spots. In some cases, chiefly on old, woody stems the spread of the lesions soon ceases, a single spot being only two or three centimeters in length by one or two centimeters broad and oval in outline. Often several spots coalesce forming a large irregular patch which may nearly girdle the stem. Within a few days the center of the spots becomes ashy white and often split or cracked, the margin being dark brown or purplish, and small raised dots appear in large numbers scattered over the surface (fig. 2b). These dots, which at first are brown and later become black, are the pycnidia of the fungus. Lesions of this type appear most often at the base of the stem and can be distinguished from the lesions of the anthracnose only by the presence of these pycnidia and the fact that the blanched center of the spot lacks the blistered appearance found in the latter disease.

In other cases, usually in succulent stems, the spots enlarge very rapidly, sometimes spreading longitudinally for a distance of several inches, and often spreading horizontally so rapidly that the stem is completely girdled within a few days. These lesions do not become white or cracked or sunken but are yellowish to dark brown, with or without a heavy dark margin, and smooth, with numerous pycnidia arranged over the surface in close concentric circles (fig. 2a). The dead host epidermis adheres closely to the stem in these brown lesions, but in the white spots on woody stems it is very loose and brittle and flakes off easily.

On the plant as a whole the effect of the blight is a general dwarfing (fig. 3b). If any blossom spikes are produced they are so weak that they are unmarketable. No lesions, however, have been observed on the flowers themselves.

## ETIOLOGY

The blight of snapdragons is caused by the pathogene *Phyllosticta antirrhini* Sydow. This fungus was originally described by P. Sydow[6] as occurring on leaves of *Antirrhinum majus* and was published by him as an hitherto undescribed species in Hedwigia in 1899. In 1900 F. C. Stewart[7] ascribed the stem disease which was first observed by him on snapdragons in a greenhouse at Geneva, New York, in 1898, to the presence of an undetermined species of Phoma.

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[6] Hedwigia 38: 134.

[7] New York (Geneva) Agr. Exp. Sta. Bul. 179: 109–110.

A comparative study of type material from Sydow's collection[8] and Stewart's original specimens,[9] together with material collected at Ithaca, New York, has shown that the morphology of the three organisms is identical. Specimens from Ithaca, sent to Stewart for examination, were pronounced by him to be affected with a fungus "without doubt" identical with that noted by him in 1900. Furthermore, as will be shown later, the fungus collected by the writer proved capable of producing all three types of symptoms (leaf spot, branch blight, and stem spot) and cultures obtained from leaves produced lesions on stems and those from stems produced lesions on leaves. From these facts it may be concluded that the fungus observed by Sydow, Stewart and the writer is the same and that it produces a disease of both stems and leaves of *Antirrhinum majus*

Attempts to discover a perfect stage of this fungus have failed so far. Old stems and leaves containing pycnidia were exposed out of doors through the winter, but repeated examinations of large amounts of material the following spring showed only pycnidia, some empty and some containing mature spores, to be present. Although the pathogene attacks both stems and leaves of the host, it seems best to retain for it the genus name Phyllosticta given by Sydow, since his was the first published description of the species.

*Pathogenicity.* By inoculation experiments with mycelium from pure cultures of the fungus, Stewart[10] practically demonstrated the pathogenicity of this organism. He inoculated snapdragon plants by puncturing the succulent shoots about three inches below the tip, inserting a bit of mycelium and covering the whole with grafting wax. In all cases the characteristic symptoms appeared, some of the branches being completely wilted within five days.

With pure cultures of the fungus, obtained by the methods described under "cultural studies" from diseased plants collected at Batavia and Ithaca. the writer has carried out additional inoculation experiments. Infections were obtained on both stems and leaves, and the fungus was reisolated from the lesions produced. Both mycelium and spore suspensions were prepared by crushing several mature pycnidia from pure cultures in a few cubic centimeters of sterilized water. In later experiments, spores from pycnidia taken directly from infected stems were used, the results being the same as those produced by spores from the pycnidia grown in pure culture.

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[8] The writer is indebted to Dr. W. G. Farlow of Harvard University and Dr. F. J. Seaver of the New York Botanical Garden for their kindness in furnishing this material.

[9] Thanks are due F. C. Stewart, Botanist of the Geneva Agricultural Experiment Station, for his generosity in loaning his original specimens.

[10] New York (Geneva) Agr. Exp. Sta. Bul. 179: 110.

For the first experiment eight vigorous greenhouse plants of different varieties, growing in 8-inch pots, were selected. Three of the plants were wounded with a sterile scalpel at different points on the main stem and on the larger and smaller branches. Before wounding, the areas had been washed with a 1-1000 solution of corrosive sublimate and then with sterilized water. A small block of agar bearing mycelium was cut from the advancing margin of a culture and smeared over the injury. The part was then covered with moist cotton and wrapped with tinfoil. Three other plants were smeared with mycelium and covered in the same manner, except that no injury was made. One of the two remaining plants was used as a check for each set, being treated in exactly the same manner as the inoculated plants except that no inoculum was used. All the plants were kept in the greenhouse where the temperature ranged from 19° to 21°C. during the day and 7° to 10°C. at night. They were examined daily, and the cotton and tinfoil were removed on the third day. In the injured plants 100 per cent infection was obtained on the green stems and about 50 per cent on the red stems, the typical water-soaked lesions appearing on the second day. The lesions spread rapidly, taking on the characteristic colors, and some of the branches were completely wilted on the ninth day. Fruiting bodies appeared in the lesions on the third or fourth day. In the uninjured stems the percentage of infection was much less, and the lesions were several days later in appearing. In the check plants no lesions appeared at any time.

In the second experiment four plants were sprayed with a spore suspension by means of an atomizer, two then being covered with bell-jars for three days and two being left uncovered. Two check-plants were sprayed with sterilized water, one being covered with a bell-jar and the other left uncovered. All the plants were kept at the same temperature as those in the first experiment. No infections were obtained in those plants left exposed to the air, but in those covered with bell-jars lesions appeared on the leaves in seven days. Pycnidia appeared almost as soon as the lesions became visible. No infections were produced on any of the stems. Both checks remained perfectly healthy.

Later experiments were tried on a large number of plants and the following results were obtained:

Leaves inoculated by a smear of mycelium and covered with moist cotton occasionally showed infections, but in the cases observed, no pycnidia were developed in the lesions. The infections on the leaves rarely spread through the blade and petiole into the stem.

Stems of all ages became infected when myceliun was used as inoculum but lesions were most frequently obtained on young shoots. On old woody stems lesions were rarely produced and were small.

Although repeated trials were made, no infections of leaves or stems either by mycelium or by spores were obtained on plants kept in a cool house where the temperature did not run above 10°C.

Spore suspensions, whether of spores from pycnidia in culture or from those in host tissue failed in every case to produce lesions on the stems. An explanation of this and the preceding point will be given later.

Cultures obtained from stem lesions produced infections in both stems and leaves and those from leaf lesions produced infections on both leaves and stems.

The above experiments prove the ability of both the mycelium and the spores of the fungus to produce the disease. Reisolations were made from the various plants becoming diseased after inoculation, and in each case the fungus obtained was identical in all respects with that in the original cultures.

*Life history. Phyllosticta antirrhini* may pass the winter as pycnidia in old lesions on the dead stems and leaves. Many shoots of the snapdragon remain green all winter, especially in places where there is some protection, such as in rubbish piles, and are only partially decayed in the spring. Partially saprophytic mycelium is abundant within such shoots, especially the succulent ones, and in the very early spring begins to form pycnidia. These pycnidia are a much more prolific source of inoculum than the overwintered ones and have been found to contain mature spores as early as the middle of April. During periods of rainfall in warm weather, the spores are discharged from the pycnidia in gelatinous coils as described below and are carried to the young plants. This form of discharge makes it probable that wind, rain and possibly insects may be the carriers of the conidia. The fact that infections occur so commonly at the tips of leaves where, due to the conformation of the snapdragon leaf, drops of water always collect after a rain, would seem to indicate that rain drops are the common carriers of spores, at least to the leaves.

In the greenhouse the pathogene may be carried over from season to season in several ways: as mycelium or pycnidia on plants brought in from the garden, in debris left in the benches, in plants moved from house to house. The spores may be disseminated by several agents, such as air currents, insects, or the hands of those who work with the plants, but probably the most important are the drops spattered about in watering the plants.

Under the proper conditions of moisture and temperature the spores germinate readily on the surface of the host and produce infections.

A detailed study of infection has been made for the leaf only, but the main features of the process are doubtless similar in the stem since the epidermal structure in the two organs is practically the same. Vaughan's[11] method of preparing material in toto proved the most useful for this work, though even with careful manipulation it did not invariably give good results. Small drops of water containing spores were placed on leaves laid on damp filter paper in moist chambers, in some cases the drops being on the upper, and in others on the lower, surface of the leaf. After being kept in a warm room for about forty-eight hours, the moist chambers were opened and the drops allowed to dry down on the leaves. The inoculated areas were then cut from the leaves and killed and fixed in Duggar's fixer (equal parts glacial acetic acid and 95 per cent alcohol). Since the cuticle of the snapdragon leaf is very thick, it was found necessary to puncture the tissue with a needle in order to secure rapid penetration of the fixer, and to soak the material in 95 per cent alcohol overnight before washing to completely decolorize it. From the 95 per cent alcohol the bits of leaf were carried back to 50 per cent alcohol, washed in several changes of the latter and then stained with Pianeze III B for fifteen to thirty minutes. When the proper reactions are obtained the pink spores and germ-tubes stand out in sharp contrast to the pale green cell walls of the host.

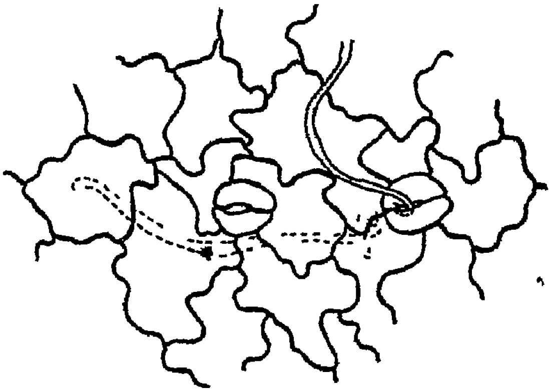

FIG. 4. TIP OF LONG GERMTUBE OF PHYLLOSTICTA ANTIRRHINI ENTERING LEAF THROUGH STOMATE AND PENETRATING EPIDERMAL CELLS

Camera lucida. × 600

The method of penetration is the same whether the germinating spore lies on the upper or on the lower surface of the leaf. The slender germ-tube grows over the surface for a considerable distance, finally turning downward at the tip and entering a stomate. The first stomate encountered is not always the one entered. The germ-tube is usually non-septate and unbranched when it enters the leaf, but several cases were observed in which branching had occurred and the tips of the branches had entered different stomates.

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[11] Vaughan, R. E. A method for the differential staining of fungous and host cells. Missouri Botanical Garden Annals 1: 241–242. 1914.

The presence of hydathodes at the tips of the leaves, offering even larger openings than the stomates for the entrance of the germ-tubes, may partly explain the frequent occurrence of lesions at the tips.

From the substomatal cavity the germ-tube enters the epidermal cells and infection is established (fig. 4).

*Morphology.* In the host tissue the mycelium is both inter- and intracellular, but no haustoria have been found. The mycelium is hyaline, irregular and stout, varying from 3 to 6$\mu$ in diameter. It is divided into many small cells by numerous septa and is profusely branched.

Sometimes simultaneously with the development of the lesions on the stems, sometimes several days later, the pycnidia become visible. On the leaves their appearance is usually delayed from one to several weeks after that of the lesion. They are imbedded in the tissue of the host, but are easily visible to the naked eye as small black papillae.

The development of these pycnidia presents no unusual features. The hyphae in the host tissues form a close gnarl just under the cuticle in the stem or just beneath the upper epidermis of the leaf, this gnarl forming the pycnidial wall made up of an outer layer of thick-walled pseudoparenchymatous tissue and an inner thin-walled layer. The mature pycnidium (fig. 5) is of the typical flask shape and protrudes slightly above the ruptured epidermis or cuticle of the host. The very short neck is made up of exceedingly heavy-walled cells, which give it the appearance, in surface view, of a black ring in contrast to the brown walls of the body. In size the mature pycnidia vary from about 56$\mu$ to 260$\mu$ in diameter, the average being about 100$\mu$. Sydow describes them as measuring 100$\mu$ to 180$\mu$ in diameter.

The cavity of the pycnidium is lined with a layer of thin-walled hyphae which stain very slightly. From this layer the minute (1 by 3$\mu$) cylindrical conidiophores arise (fig. 5). The small hyaline pycnospores are cut off from the conidiophores by constriction. They are produced in great numbers and are imbedded in a gelatinous matrix, the whole mass completely filling the cavity of the pycnidium. In the presence of moisture the gelatinous matter swells and the spores are extruded from the ostiolum, held together in a long whitish coil. Some idea of the large number of spores formed in a single pycnidium may be gained from the fact that the complete emptying of a pycnidium of average size was observed to require twenty-three minutes, the coil of spores attaining a length of over 8 cm.

The pycnospores (fig. 6) are cylindrical with blunt rounded ends to nearly ellipsoid. They are biguttulate, hyaline, 4 to 6 by 1 to 2$\mu$ (usually 4 by 2$\mu$), (4 to 6 by 1.5 to 2$\mu$, Sydow).

When placed in tap water, distilled water, or snapdragon leaf decoction or on potato agar, and kept in a warm place, the spores from mature pycnidia germinate within eighteen to twenty-four hours. Spores placed in drops of water and on very thin films of potato agar on the inverted covers of van Tieghem cells, and kept from 23° to 29°C., gave nearly 100 per cent germination in about eighteen hours, growth of the hyphae being much more profuse in the agar film.

At temperatures lower than 18°C. or higher than 47°C. or in the absence of a film of water germination does not occur. Spores placed on dry covers of van Tieghem cells, or in drops of water so small that they soon dried off, failed to germinate, although the air of the cell presumably was kept moist by the large drop of water in the bottom. Spores kept at a temperature of 47°C. or higher and 18°C. or below did not germinate at all, while those kept at 20° to 21°C. gave a small percentage of germination, the germ-tubes, however, not developing into mycelium.

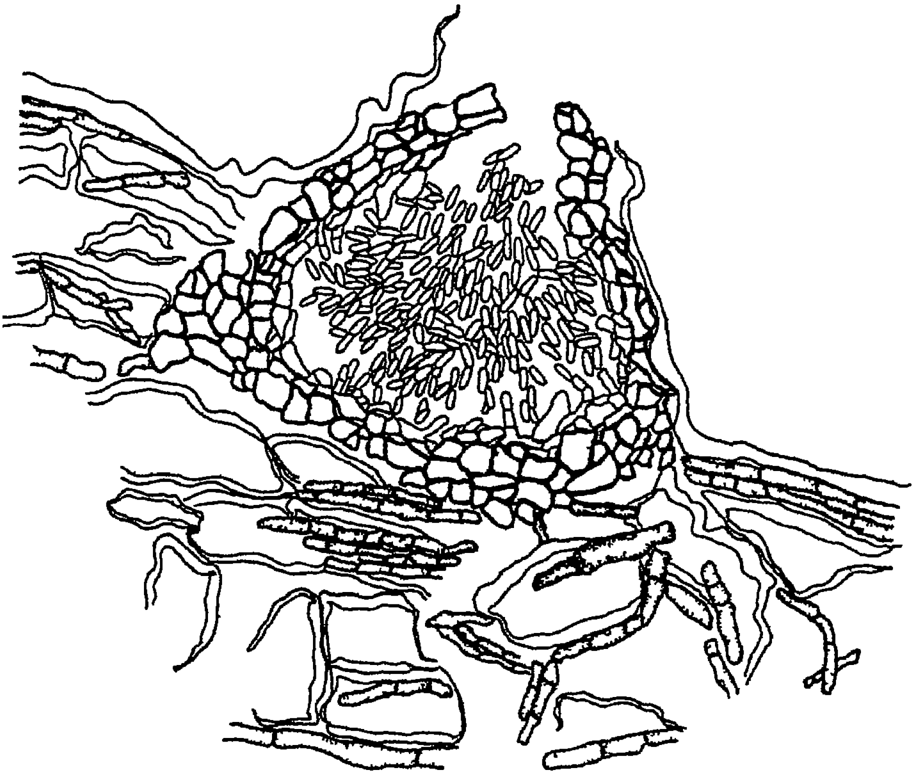

FIG. 5. MATURE PYCNIDIUM OF PHYLLOSTICTA ANTIRRHINI IN CORTEX OF HOST STEM

Longisection. Most of spores have been extruded. Camera Lucida. × 600.

The first evidence of germination of the spore is a marked swelling, followed by the appearance at the end of the spore of a stout germtube, almost as large in diameter as the spore itself. One or two germtubes are sent out from one end of the spore, or a tube emerges from each end (fig. 6). The germ-tubes elongate rapidly and become septate, soon branching to form the mycelium.

A consideration of the factors governing germination in the above tests may explain the negative results obtained in certain of the inoculation experiments. No infection was produced when plants inoculated with a spore suspension were kept in a house where the temperature did not rise above 10°C. Since a temperature of 20°C. or higher is most favorable for spore germination, infection probably failed because no germination occurred. The host stems of *Antirrhinum majus* are provided with a heavy cuticle and so shed water and dry off rapidly. Thus it is possible that in the case of the stems sprayed with a spore suspension, although the plants were kept in a warm house, infection failed because the drops of water containing spores did not adhere long enough for germination to take place. If any of the spores adhered, the necessary film of water may have been lacking, preventing germination in this way.

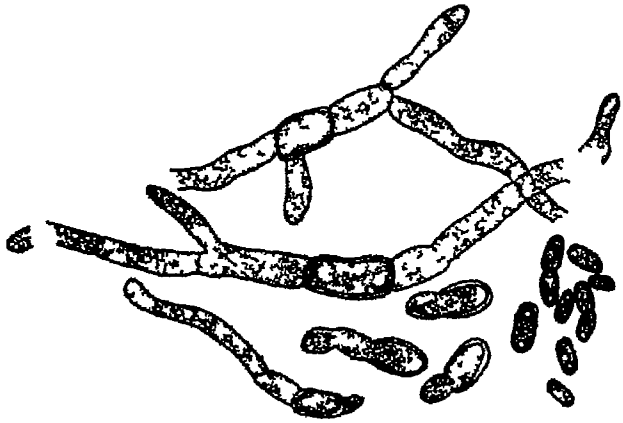

FIG 6. MATURE AND GERMINATING PYCNOSPORES OF PHYLLOSTICTA ANTIRRHINI Note swelling at germination. Camera lucida. × 625

CULTURAL STUDIES

Pure cultures of *Phyllosticta antirrhini* were repeatedly obtained in the following manner:

Pieces of diseased stems or leaves were disinfected on the surface by immersing in 95 per cent alcohol for several seconds, then soaking in 1–1000 corrosive sublimate solution about one minute. They were rinsed in five changes of sterilized water and bits of the infected tissue were then taken out with sterilized forceps and transferred to sterilized media.

In obtaining pure lines of the fungus, single hyphal tips were cut from the margin of vigorously growing young cultures by means of a sterile sharp pointed scalpel and transferred to new media. This method was found to be more satisfactory than the single spore method on account of the difficulty of locating the small hyaline spores in a poured plate.

Observations on the cultural characters of the fungus were made with only the more common solid media: potato, oatmeal, bean, cornmeal and nutrient agars, bean plugs, and with beef bouillon. Some marked differences in the growth on the various solid media were noted.

On nutrient agar the growth of *Phyllosticta antirrhini* is very slow and weak, and fruiting bodies are not produced.

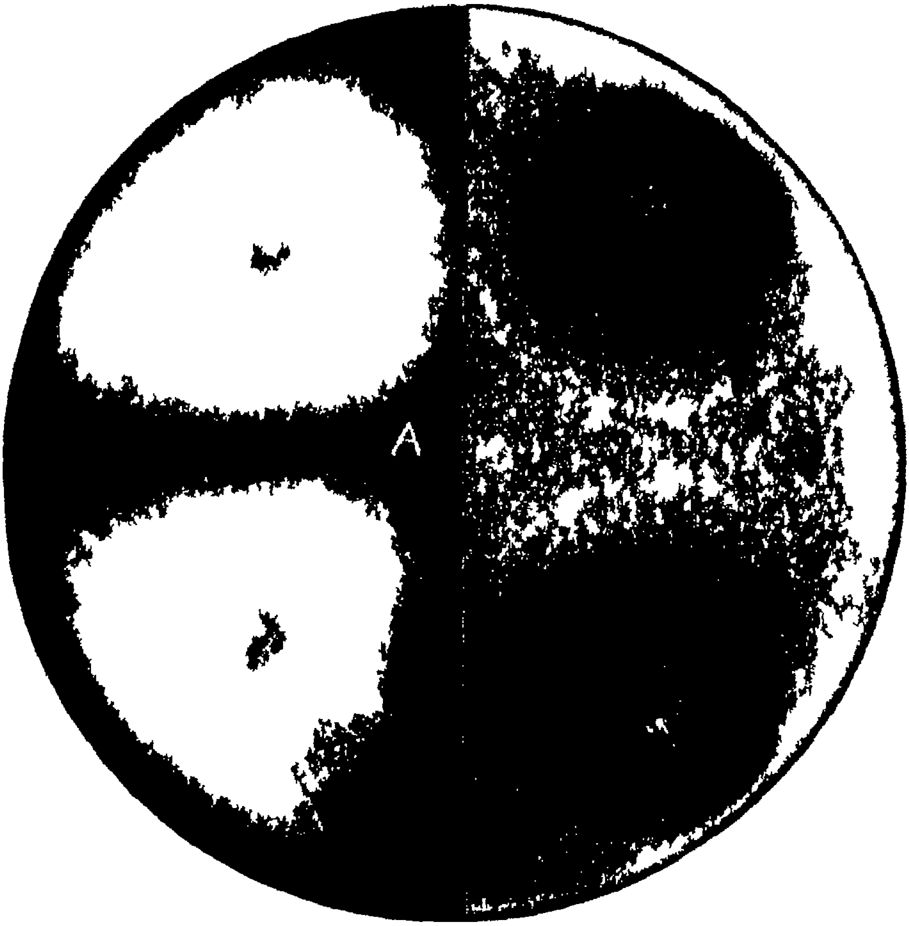

FIG. 7. GROWTH OF PHYLLOSTICTA ANTIRRHINI ON POTATO AND CORNMEAL AGAR. FIVE DAYS.

*A*. Yellowish white growth on potato agar. *B*. Greenish black growth on corn meal agar.

The growth of the hyphae in beef bouillon is very rapid. The medium is permeated by mycelium in several days and a heavy yellowish grey pellicle is formed at the surface. No fruiting bodies are formed in this medium.

In potato and bean agar the growth of the fungus is rapid and profuse, becoming visible macroscopically within twenty-four hours after planting

The typical growths on these media are low (aerial hyphal branches not more than 0.5 mm. high) and thin, the hyphae penetrating the substratum only slightly. In color the growths are yellowish and smooth below, sordid white and floccose above, with a nearly colorless zone at the extreme margin. The pycnidia appear early, usually within two or three days after the mycelium becomes visible. They are at first brownish, then become black, and are scattered in concentric circles around the oldest part of the planting, soon becoming abundant and dotting the whole surface. Older cultures (about eight days) are distinctly zonate. In cultures several months old, the early growth is overgrown by a thick, black, stroma-like crust which is glossy, except where a scanty white mycelium still persists. Pycnidia formed in this crust are larger but less abundant than those produced in the young, vigorously growing cultures.

On oatmeal and cornmeal agar the growth is similar in form to that on the potato or bean agar, but is strikingly different in color. As early as the third day after planting the growth is greenish black, with a more or less glossy surface and with a colorless band at the extreme margin. The color is located in the mycelium itself, and is not due to a discoloration of the medium. Pycnidia are produced as abundantly as in potato and bean agar, and the same zonate character appears in cultures five to seven days old. Old cultures (three to four weeks) are uniformly dull-black, the zonate character having disappeared.

The typical young cultures on the two types of agar are shown in comparison in figure 7.

## PATHOLOGICAL HISTOLOGY

In both stems and leaves the lesions produced by this disease are necrotic. The mycelium is inter- and intracellular as already stated. In the stems it is found beneath the cuticle and between the cells in long strands made up of several parallel hyphae, while within the cells single hyphae pass from lumen to lumen. Apparently the mycelium is able to penetrate the walls of the softer cells directly but must enter the thicker walled cells such as the tracheids through the pits. All types of tissue with the possible exception of sclerenchyma are attacked. Vessels are rarely invaded. The epidermal cells are the first to be invaded, the mycelium spreading from them to the outer cortical cells, then progressing more deeply into the stem through the phloem and cambium regions to the xylem and pith. Injury to parenchymatous cells is apparent several cells in advance of the mycelium. The contents of such cells are completely destroyed. They are first broken down, then disappear entirely. The empty walls soon collapse leaving the tissue merely a mass of broken walls and tangled hyphae. The presence of the hyphae produces no apparent effect on the invaded lignified cells (fig. 8).

The histological changes which occur in the leaf are similar to those seen in the stem. The same destruction of the soft tissues takes place, while the veins are penetrated but remain intact. The mycelium formed from the germ-tube attacks first the epidermal cells, spreading in all directions from the point of penetration. The epidermal cells are killed rapidly, and the palisade cells are next attacked. These cells soon die and the infection spreads downward through the leaf until finally all tissues are involved.

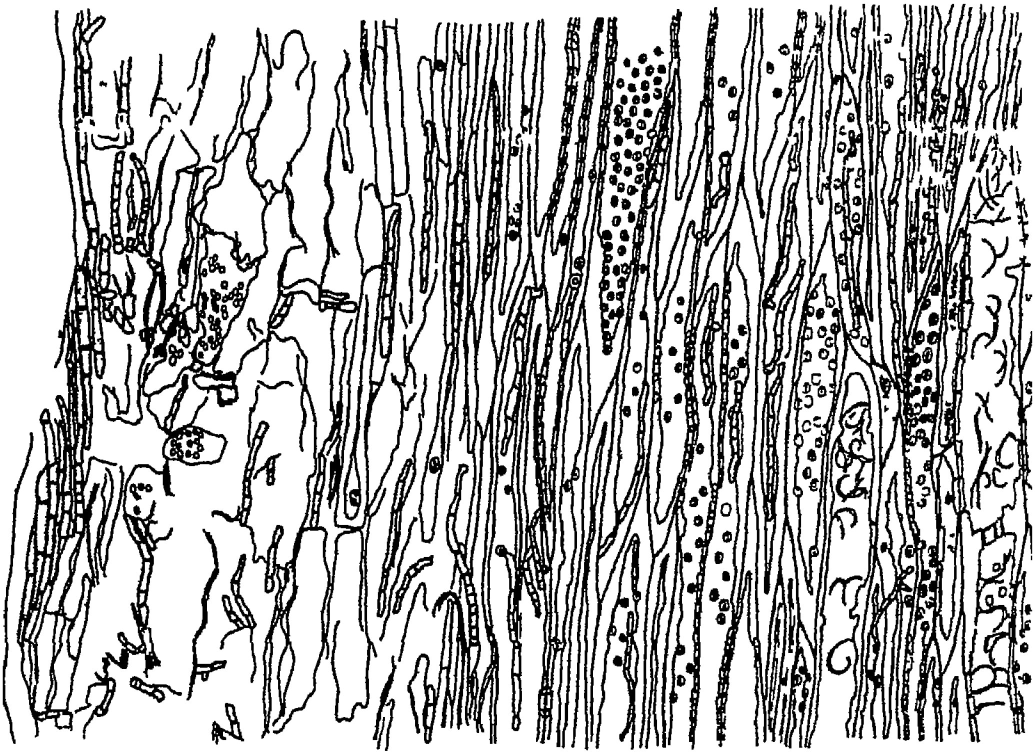

FIG. 8. PATHOLOGICAL EFFECT OF PHYLLOSTICTA ANTIRRHINI ON TISSUES OF YOUNG SNAPDRAGON STEM

Note disintegration of cortex, phloem and cambium regions and invasion of xylem elements. The mycelium is seen passing through the pits of the xylem cells. Camera lucida. × 202.

The successive stages in the disintegration of the cells of both leaves and stems are easily studied in microtome sections cut about 5μ thick. Satisfactory results are obtained in staining such material with either Flemming's triple stain or Heidenhain's iron alum-haematoxylin.

The cytoplasm is the first to show the effects of the presence of the fungus. Before the cell wall is penetrated by the hyphae, the cytoplasm, probably attacked by some toxin or enzym secreted by the advancing mycelium, breaks up into coarse granular particles and finally disappears altogether. The nucleus remains visible for a time but soon breaks up and disappears in its turn. The chlorophyll bodies, when present, are the last to disappear, and may often be found apparently intact for some time after the walls are empty of other content. At length they too disappear. The cell walls may collapse soon after the breaking up of the cytoplasm, or they may remain normal for a time after all contents are destroyed, even though penetrated by hyphae. Eventually, however, they collapse and become more or less broken.

The line of demarcation between the healthy and diseased tissues is very sharp, but no evidence of cork formation or of suberization could be discovered.

The collapse of all soft tissues in both stems and leaves is responsible for the sunken appearance of the lesions, and the complete breaking down of the cells and drying of the tissues causes the brittleness and ready crumbling of the affected areas described under symptoms.

## CONTROL

Little work has as yet been done on the control of the Phyllosticta blight. Stewart[12] offers the suggestion that spraying with Bordeaux mixture as for anthracnose may be efficacious. It seems probable also that dusting with sulfur would control this blight. This method would give the additional advantage of controlling the rust at the same time. The planting of snapdragons in gardens where the disease was prevalent the preceding year should be avoided unless all debris can be removed.

From the observations of the conditions required for spore germination and from the results obtained in the inoculation experiments, it seems possible that in greenhouse plants the disease can be greatly diminished if not entirely controlled by certain precautionary measures. The first is of course the removal of all debris from infested benches before new plants are put in and the use of only healthy plants for setting. The second precaution is the practice of soil watering, with proper ventilation of the house. Finally, snapdragons should be grown in cool houses, for the plants will do well in an average temperature of 15°C., a temperature at which the fungus can not thrive as a parasite.

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[12] New York (Geneva) Agr. Exp. Sta. Bul. 179: 110.

# PHOMOPSIS JUNIPEROVORA, A NEW SPECIES CAUSING BLIGHT OF NURSERY CEDARS

GLENN GARDNER HAHN

WITH ONE FIGURE IN THE TEXT

In the writer's early studies of the nursery blight of cedars (3) an undetermined organism was isolated from *Juniperus virginiana* L. and found to be the causal fungus. At that time this fungus was referred temporarily to the genus Phoma, until determination work of a more complete nature had been accomplished.

By growing the fungus secured from a series of isolated spores, in pure culture upon corn meal agar and prune agar in petri dishes, and steamed rice and corn meal in flasks, fruiting bodies were secured, but these in no case observed were of an ascigerous stage. Sections of the pycnidia produced by the inoculation of *J. virginiana* in the greenhouse showed abundant occurrence of scolecospores (The A and β type spores of Diedicke (1)) characteristic of the genus Phomopsis (2). Field material which previously had shown apparently only A type spores and what appeared to be a Phlyctaena, upon further sectioning and examination showed β type spores occurring in the same pycnidium with the A spores. This observation of the two spore types, together with the observation as to the nature of the pycnidium (which, as was earlier indicated, did not refer the fungus to a typical Phoma), definitely shows the organism to belong to the genus Phomopsis, and inasmuch as it differs from all other fungi so far described as infectious to *J. virginiana* and from all other Phomopses upon conifers, it is now described as *Phomopsis juniperovora* n.sp., having the following characteristics:

## Phomopsis juniperovora n.sp. (fig. 1, A)

Pycnidiis sparsis, fuscis, carbonaceis, lenticularibus, conoideis, vel truncatis bis subglobosis; initio subepidermicis denique erumpentibus, astromatoideis, ostiolatis vel non definite ostiolatis, contextu heterogeneo praeditis, matricibus saepe supra cavitates sporiferas incrassatulis; in foliis,—epiginis, raro hypogenis, lenticularibus bis subglobosis, unilocularibus, 124–310μ diameter, vulgo 200μ; in truncis—conoideis, truncatis vel lenticularibus, basi latis, depressis, unilocularibus, matricibus proliferis frequenter dissseptimenta formantibus; 186–620μ diameter vulgo 350μ.

Sporulis ellipsoideis, oblongis vel fusiformibus hyalinis, continuis, biguttulatis, 6.5 to 12 by 1.8 to 3.5μ, vulgo 7.5 to 10 by 2.5 to 3μ. Basidiis filiformibus, attenuatis, con-

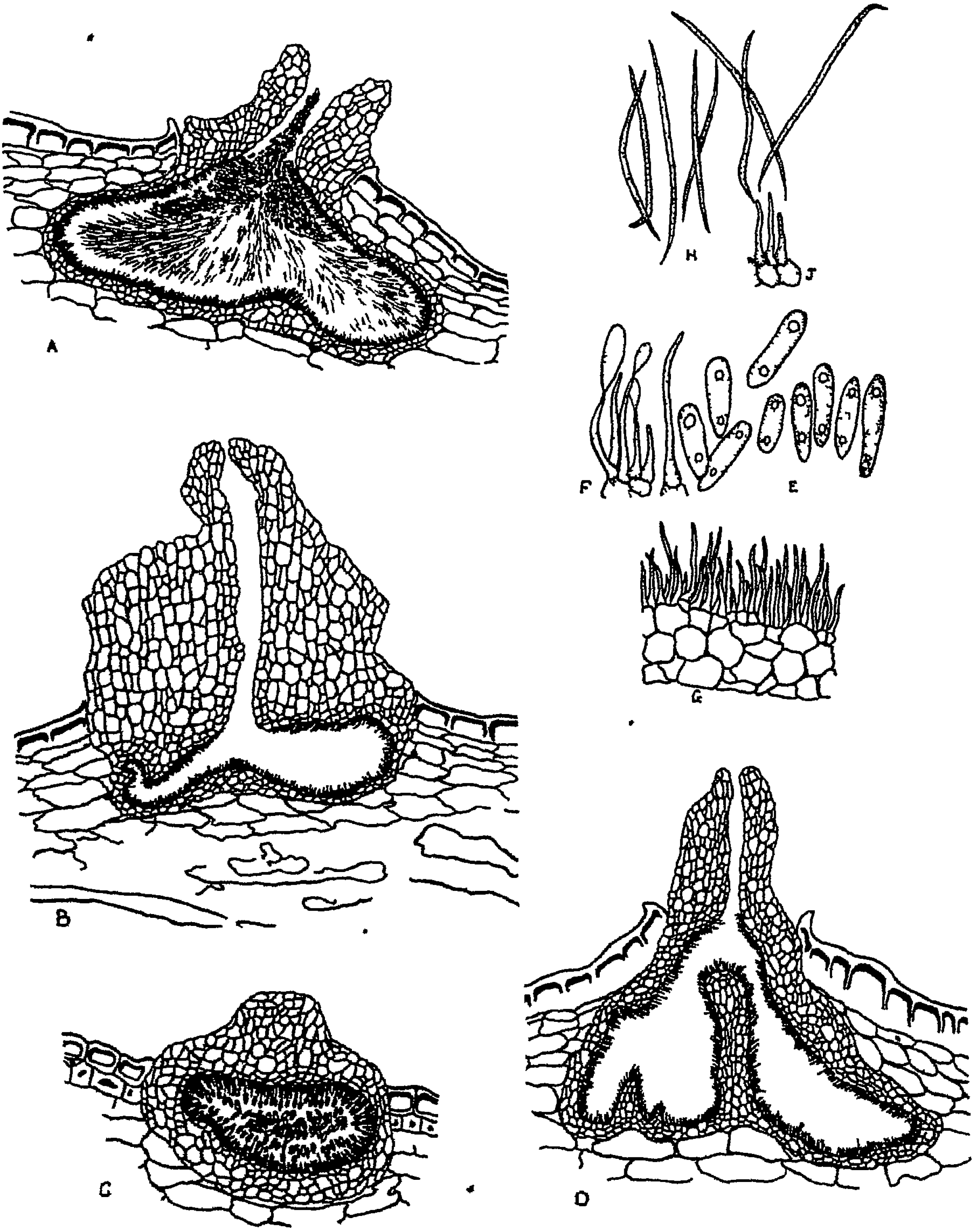

FIG. 1

*A.* A typical pycnidium of *Phomopsis juniperovora* upon the stem, showing both A and B type spores. Drawn with the aid of the camera lucida. Approximately 113 X.

*B.* Pycnidium upon the stem, showing the dense pseudoparenchymous layer overlying the spore cavity. It is this growth which breaks through the epidermis, frequently before spores are formed. The chimney-like ostiole is characteristic. Drawn with the aid of the camera lucida. Approximately 113 X.

tinuis, subulatis, 5 to 15 by 1 to 2μ Scolecosporulis filiformibus, rectis, vel leviter curvulis, vel hamatis, raro sigmoideis, 17.5 to 28 by 1μ, vulgo 20 to 24 by 1μ. Basidiis brevibus, subulatis, 5μ

Hab.—In acibus et truncis Juniperi virginianae, Dundee, Ill. 1916.

Pycnidia lens-shaped, conical, truncate, at times subglobose typically depressed with a broad base; embedded at first but becoming erumpent by the upper part of the pycnidium breaking through the epidermis before or with maturity; separate; without a stroma; texture, heterogeneous; the spore-bearing cavity floored below by a layer of faintly colored cells, two to several thick, and roofed above with a denser layer, at times exceedingly thick; (fig. 1, *B*); toward the outside the cells may be considerably darker, brown or black, ostiolate but generally provided with an opening slit or irregular orifice; upon the leaves, lens shaped to subglobose (fig. 1, *C*), epigenus, always simple chambered, 124 to 310μ diameter, average 200μ; upon the stem, cone shaped, truncate or lens shaped, simple chambered, frequently with rudimentary partitions arising from the proliferous layer, usually from the lower floor or base of the cavity (fig. 1, *D*), 186 to 620μ diameter, average 350μ. Spores oblong to ellipsoid, or spindle shape, with subacute extremities, usually biguttulate, occasionally with more than two oil drops, hyaline, continuous, 6.5μ to 12 μ by 1.8 to 3.5μ (100 measurements) (fig. 1, *E*); commonly 7.5 to 10μ by 2.5 to 3μ; sporophores filiform, linear, subulate, often slightly curved, 5 to 15 by 1 to 2μ (fig. 1, *F*) in close set, compact layers, persistent (fig. 1, *G*).

Associated with the A spores and produced in the same pycnidium are β spores or scolecospores. These may also occur alone in fruiting bodies approximating the Phlyctaena genus type. The second spore forms are long, filamentous, occasionally curved S-shaped, or hooked at the upper end, 17.5 to 28μ by 1μ, generally 20 to 24μ by 1μ (fig. 1, *H*). Sporophores short subulate, about 5μ (fig. 1, *J*).

Spores extruded in a cream colored, gelatinous tendril 1 to 5 mm. long, becoming brittle upon drying.

Type specimen in the Pathological Collections, B. P. I., U. S. Department of Agriculture, and Office of Investigations in Forest Pathology (F.

*C*. Pycnidium upon a leaf. Drawn with the aid of the camera lucida. Approximately 197 ×.

*D*. Pycnidium upon the stem showing false partitions arising from the floor of the spore-bearing cavity. Drawn with the aid of the camera lucida. Approximately 113 ×.

*E*. Typical A type spores 1125 ×.

*F*. Sporophores of A type spores. 1125 ×.

*G*. Proliferous layer and sporophores. 563 ×.

*H*. B type, or scolecospores. 1125 ×.

*J*. Sporophores of B type spores. 1125 ×.

P. Nos. 25002, 25005) on diseased *Juniperus virginiana* L., nursery grown stock, three years old, from Dundee, Ill., June 1916.

*Distribution.* Nebraska (3); nursery stock of *J. virginiana* L. in Kansas, Illinois, Minnesota, Iowa,[1] Ohio, New York, Pennsylvania.

## CULTURAL CHARACTERISTICS

The cedar blight organism grows well on corn meal agar, prune agar, steamed rice, and steamed corn meal. It fruits more frequently on the corn meal agar and on the corn meal flasks, than on the other media mentioned.

Growth of the fungus is silky, appressed on corn meal agar in petri dishes, with or without a slight tendency to zonate, at first empire yellow (4), becoming orange rufous after twenty days. The pigment is diffused throughout the medium, in a radius of 10 mm. beyond the extending edge of the colony. This is accompanied by the formation of flaming orange crystals, which may occur within twelve days. These crystals are observed less abundantly on prune agar. Here color pigmentation passes through the same shades during the early development of the colony, but finally becomes tawny. Growth characteristics very much resemble those upon corn meal agar.

## COMPARISON WITH OTHER SPECIES OF PHOMOPSIS ON CONIFERS

In the literature exceedingly few species of Phomopsis have been described on conifers, although undoubtedly others will be found which at the present time are described under other genera. A Phomopsis occurs on *Thuja occidentalis* L. in Europe and has been described by Diedicke as *P. thujae* Died. n. sp.[2] This fungus very much resembles the *P. juniperovora* in that it apparently has the same morphological features as to the nature of its pycnidium. The A spores, however, differ in that the foreign species has a smaller spore, 5 to 8 by 2$\mu$. The *P. juniperovora* spores are longer and wider. Unfortunately specimens of the *P. thujae* in the Pathological collections, U. S. Department of Agriculure, distributed as No. 1018 Exsiccatus Sydow Mycotheca germanicus[3] proved to be only a species of Diplodia, so exact comparison was impossible. A note accompanying the foreign specimens states that the *P. thujae* is frequently associated with a mature Diplodia. Evidently only Diplodia material was sent over.

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[1] Specimens sent from a nursery at Kent, Iowa, (1896), to Dr. F. C. Stewart, N. Y. (State) Exp. Station (Phytopathology, 8: no. 1. Jan., 1918). Later, collections of material were also received by the Office of Forest Pathology, B. P. I., U. S. Dept. Agr., from this State (1916).

[2] Kryptogamenflora der Mark. Brandenberg, 9: 275. 1912-1915.

[3] Annales Mycologici. 9: 557. 1911.

It is here interesting to note that the parasitism of *P. juniperovora* has been proven positively upon young nursery trees, in the greenhouse, of *Thuja occidentalis* and *T. orientalis* L. At the same time inoculations upon three pines, *Pinus edulis* Engelmn., *P. glabra* Walt., and *P. taeda* Linn., gave negative results, as well as upon *Pseudotsuga taxifolia* (Lam.) Britton, *Picea parryana* (André) Parry, *P. engelmanni* Engelmn., and *Abies lasiocarpa* (Hook) Nutt.

That the *Phomopsis juniperovora*, now a widely known distributed parasite upon young nursery cedars, particularly red cedar, is distinct from the European fungus of Diedicke is the opinion held; however, it is interesting here to call attention to a possible similarity between the two. The identity of the *Phomopsis thujae* Died. with the native Phomopsis on nursery cedars cannot be definitely established until a direct morphological comparison has been made from type material, and cross inoculation has been performed to prove parasitism.

OFFICE OF INVESTIGATIONS IN FOREST PATHOLOGY
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# PHYTOPATHOLOGICAL NOTES

*Annual general meeting of the Association of Economic Biologists.* Mr. W. B. Brierley, Botanical Secretary, has furnished a brief report of the annual meeting of the Association of Economic Biologists, which was held in the Imperial College of Science on December 10 and 11, 1919. Over seventy new members joined the association. Sir David Prain, C.I.E., C.M.G., F.R.S., was elected president for the ensuing year.

Wednesday morning was devoted to short communications and exhibitions, upon each of which there was an active discussion. Mr. E. E. Green was in the chair. Mr. A. D. Cotton showed a collection of very beautiful photographs of tree diseases prevalent in the United States. Mr. W. F. Bewley showed tomato diseases destructive in the Lea Valley glass houses. A Tingid bug, attacking Rhododendrons, was exhibited by Mr. E. E. Green; whilst Mr. W. B. Brierley described a growth of *Citromyces pfefferianus* in pure deci-normal sulphuric acid, which had been sent to Rothamsted by Dr. Golding of Reading.

On Wednesday afternoon the following papers were read and discussed, Mr. A. G. L. Rogers occupying the chair: Mr. W. F. Bewley on "Sleepy Disease or Wilt of Tomato." The pathogen has been named *Fusarium lycopersici*, but this is composed of two discrete fungi, a parasitic *Verticillium* sp., and a saprophytic *Fusarium* sp., the former being the so-called *Diplocladium* stage. The pathogenicity of the *Verticillium* sp. is conditioned by a temperature factor, and there are various strains differing constantly in physiological properties.

Mr. W. E. Hiley on "A new Instrument for Measuring the Light Intensity in Woods." Solutions of chlorophyll and pure spirit are connected by a copper-constantin thermocouple and a current of about 1–3 by $10^{-5}$ amperes obtained, which on a sensitive galvanometer gives a deflection of two inches. The instrument will be used in measuring the light intensity necessary for the growth of shade-bearing trees used for underplanting light demanders, and to test the value of forest weeds as indicators.

Mr. F. R. Petherbridge on "The Life History of the Strawberry tortrix, *Acalla comariana.*" The life history of the tortrix, its distribution, and the damage it causes, were described, and remedial treatment indicated.

Thursday was devoted to a symposium on "The integration of mycological research with practice in Agriculture, Horticulture, and Forestry." Dr. F. Keeble occupied the chair.

Sir Daniel Hall discussed the Administrative problem. The relation of "free" to "administrative" research was dealt with, the former being controlled by a University or kindred body and only remotely subject to government criticism; the latter being an integral part of the government's activities. The advantages and disadvantages of directed team work were discussed and administrative difficulties in this connection indicated. In dealing with plant diseases the Board of Agriculture proposes the following organization:—

(I) The institutions engaged in free research.

(II) The Board's own staff of advisors and inspectors dealing with administrative research.

(III) A corps of local investigators attached to the agricultural colleges.

It is anticipated that it will be possible through the Councils of Research and Advice to keep these bodies so far in touch that from time to time combined investigations can be undertaken of particular widespread and pressing problems.

Prof. V. H. Blackman discussed the training problem. Emphasis was laid on the fact that the prospects of a career must be sufficiently satisfactory to attract students of high ability. A wide outlook is essential in this work and students must not only have a good general knowledge of physics and chemistry, but a thorough grounding in botany and special training in mycology, mycological technique and plant pathology. No attempt should be made to produce a fungal systematist. Acquaintance with the results of plant breeding is advisable, as well as of medical bacteriology and entomology. The physiological outlook is essential and field experience very important.

Dr. E. G. Russell discussed the agricultural problem. Emphasis was laid on field study of the growing plant in health and in disease, both states being regarded as the resultant of the prevailing conditions. The extreme complexity of the problems was pointed out and illustrations given of the ways in which they are being attacked at Rothamsted. The relations between the central research station for fundamental investigations and the small research sub-station devoted to particular aspects of crop production was indicated. The special difficulties of mycological research on agricultural crops were dealt with, together with the best methods of approach to the general problems, and it was concluded that in essential principles the work was more in common with that done among human beings on public health and eugenics than with curative medicine.

Mr. F. J. Chittenden discussed the horticultural problem. The main problems confronting the mycologist in horticultural practice were indicated and the special difficulties described. These largely result from the intensive methods used, the vast variety of plants grown, many of very limited adaptability, the controlled conditions of cultivation, and the transference of plants from place to place. The task of the mycologist is not only to devise such methods of control as are practicable under the conditions imposed, but to bring these home to the practical man. Emphasis was laid on the value of demonstration gardens as a means of education.

Prof. W. Somerville discussed the forestry problem. The peculiar difficulties of mycological research in relation to forestry arise in the fact that in practice the returns are long deferred, the annual value of the output per acre is relatively small, the operations are conducted in less accessible areas, and the crop (trees) is less amenable to treatment. The possibility of breeding immune varieties and the difficulties involved were indicated. Emphasis was laid on the necessity for pure research, but the diffusion of its results among practical foresters is not easy, owing to the obstacles involved in testing conclusions on a crop which may not mature for a century. The importance of the present widespread changes in land ownership and the bearing of the functions of the newly created Forestry Commission upon the new conditions introduced, was dealt with. It was concluded that it is in the improvement of education all round that we may most hopefully look for continued appreciation of science and that when this advance has been secured the integration of research with practice will be a natural and inevitable consequence.

Sir David Prain briefly described the functions of the Imperial Bureau of Mycology, now being formed, and its relation to existing institutions.

The general discussion in the afternoon was opened by Mr. E. S. Salmon, and the following took part: Messrs. Brooks, Cotton, Brierley, Henry, Bruce, Taylor, Lister, Gough, Bewley, Willis, Ramsbottom, Wormald, Paine, Keeble, and the Misses Cayley and Wakefield.

*Check list of Department of Agriculture publications.* There are still on hand available for distribution copies of the "Check List of publications of the Department of Agriculture on the subject of Plant Pathology, 1837–1918," which was issued in June, 1919, as Bibliographical Contributions No. 1 of the U. S. Department of Agriculture Library. Copies may be procured by application to the Library, U. S. Department of Agriculture, Washington, D. C.

*Personals.* Dr. L. O. Kunkel resigned the position of plant pathologist in the U. S. Department of Agriculture, on February first, to become associate pathologist of the Hawaiian Sugar Planters' Experiment Station, Honolulu. He will be engaged in work on the mosaic disease of sugar cane.

Dr. B. O. Dodge, for several years a member of the Department of Botany of Columbia University, has accepted a position as plant pathologist in the office of Fruit Disease Investigations, U. S. Department of Agriculture.

Mr. Charles R. Hursh has resigned his position as assistant pathologist in Cereal Disease Investigations to take up graduate study at the Missouri Botanical Gardens.

Dr. E. Mead Wilcox has resigned as professor of plant pathology in the University of Nebraska and plant pathologist of the Experiment Station, effective April 1, 1920, to accept the directorship of the Agricultural Experiment Station being established at Santo Domingo in the Dominican Republic.

*Washington meeting of the Advisory Board of American Plant Pathologists.* The Advisory Board met in Washington January 29 to 31 with the following commissioners present: C. R. Orton, L. R. Jones, E. M. Freeman, H. S. Jackson, A. H. R. Buller, and G. R. Lyman. The first session was held at the headquarters of the National Research Council on the invitation of Dr. C. E. McClung, Chairman of the Division of Biology and Agriculture. The Advisory Board is the committee on Phytopathology of the Research Council and this meeting not only afforded an opportunity for the Board to consider important matters referred to it by the Council, but also enabled the members of the Board to become personally acquainted with the officers and work of the Council.

Detailed consideration was given to the Phytopathological Institute in consultation with officers of the National Research Council. Dr. Freeman, Chairman of the committee on securing funds, spoke on the proposed Institute at a special meeting of the Pathological Seminar of the Bureau of Plant Industry. It is felt that definite progress is being made in developing a wide-spread interest in the Institute and that the prospects are good for its realization in the not distant future.

The status of the various coöperative projects of the Board was considered. There is imperative need for funds to permit the leaders of these projects to push this work and to properly correlate the results obtained. That American plant pathologists have the spirit of coöperation is clearly shown by the discussions at the St. Louis meeting, but the effective prosecution of coöperative projects requires funds to permit conferences of the coöperators, the securing of uniform material, and the comparison of results. It is hoped that such funds can be secured, either through the agency of the National Research Council, or independently. In accordance with the vote of The American Phytopathological Society at St. Louis the Advisory Board took steps to inaugurate coöperation on dusting as a means of plant disease control. Dr. N. J. Giddings of the West Virginia Experiment Station was requested to assume leadership and those interested in this project should communicate with him.

One of the most important topics before the Advisory Board at this meeting was the consideration of plans for a summer field meeting during 1920. It was determined to hold such a meeting about the first week in August with particular reference to fruit diseases. The tentative itinerary decided upon involves assembling at some point in the Shenandoah Valley, probably at Winchester, Va., whence passage will be made by automobile northward through West Virginia and Maryland to the Gettysburg section of Pennsylvania. The duration of the conference will be three or four days. The itinerary contemplated covers one of the most attractive sections in the eastern United States,—agriculturally, historically and scenically. Careful consideration was given to plans for inviting pathologists from abroad to visit the United States at the time of this field meeting. This matter was finally left in the hands of a committee consisting of G. R. Lyman, C. L. Shear, and W. A. Orton. The following committee on arrangements was appointed to have charge of perfecting plans and making all arrangements for the Shenandoah Valley conference: F. D. Fromme, chairman, representing Virginia; N. J. Giddings, representing West Virginia; C. E. Temple, representing Maryland; C. R. Orton, representing Pennsylvania; M. B. Waite, representing The Bureau of Plant Industry; G. R. Lyman, representing the Advisory Board.

G. R. LYMAN,
*Chairman.*

# LITERATURE ON PLANT DISEASES[1]

COMPILED BY ALICE C. ATWOOD, AND FLORENCE P. SMITH, BIBLIOGRAPHICAL ASSISTANTS, BUREAU OF PLANT INDUSTRY

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All authors are urged to cooperate in making the list complete by sending their separates and by making corrections and additions, and especially by calling attention to meritorious articles published outside of regular journals. Reprints or correspondence should be addressed to Miss E. R. Oberly, Librarian, Bureau of Plant Industry, U. S. Dept. of Agric., Washington, D. C.

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# APPENDIX

## REPORT OF THE ELEVENTH ANNUAL MEETING OF THE AMERICAN PHYTOPATHOLOGICAL SOCIETY

The eleventh annual meeting of the Society was held in Soldan High School, St. Louis, Missouri, December 29, 1919, to January 2, 1920, in conjunction with the American Association for the Advancement of Science and the Botanical Society of America The Society headquarters were at Marquette Hotel.

About ninety members were present. One hundred and forty-four new members were elected, making the total active membership of the Society five hundred and twelve on January 1, 1920.

Forty-nine papers were presented at the regular sessions. Abstracts of these appeared in the January number of PHYTOPATHOLOGY. One session was devoted to consideration of the topic "Eradication of Plant Diseases: Is it Practicable, and when should it be Attempted?" Certain phases of this question were presented briefly by selected speakers, after which there was general discussion.

Three sessions were held jointly with other societies. The first of these was held Tuesday afternoon, December 30, with Section G of the American Association for the Advancement of Science and the Botanical Society of America. At this meeting the address of the vice president of Section G was presented, and also invitation papers on phylogeny of seed plants. The second session was held Thursday afternoon, January 1, with the Botanical Society of America, for the presentation of papers on mycology and plant pathology. The third session was held Wednesday afternoon, December 31, with the Botanical Society of America and the American Society for Horticultural Science. By invitation this last session was held at the Missouri Botanical Garden. A tour of inspection through the Garden and luncheon in the laboratory building preceded the program of invitation papers.

The annual dinner of the Society was held Tuesday evening, December 30, at the American Hotel Annex, and was attended by one hundred and seven phytopathologists and botanists. The dinner was followed by an extended discussion of the proposed Phytopathological Institute.

The following officers were elected:

*President*, Dr. Wm. A. Orton, United States Department of Agriculture, Washington, D. C.

*Vice president*, Dr. E. C. Stakman, University of Minnesota, University Farm, St. Paul, Minnesota.

*Councilor for two years*, Dr. G. H. Coons, Michigan Agricultural College, East Lansing, Michigan.

*Members of editorial board:* Editor elected for two years, Prof. H. S. Jackson, Purdue University, Lafayette, Indiana, and editor elected for three years, Dr. G. M. Reed, United States Department of Agriculture; associate editors for three years, Dr. N. J. Giddings, University of West Virginia, Morgantown, W. Va.; Dr. Neil E. Stevens, United States Department of Agriculture, Washington, D. C.; Dr. G. H. Coons, Michigan Agricultural College, East Lansing, Mich.; and Dr. Perley Spaulding, United States Department of Agriculture, Washington, D. C.; and business manager elected for one year, Dr. G. R. Lyman, United States Department of Agriculture, Washington, D. C.

*Member of the Editorial Committee of the American Journal of Botany for three years*, Dr. C. L. Shear, United States Department of Agriculture, Washington, D. C.

The Society voted to hold its next annual meeting at Chicago, Ill., in conjunction with the American Association for the Advancement of Science, December 27, 1920, to January 1, 1921, inclusive.

### AMENDMENT TO THE CONSTITUTION

The following amendment to the constitution, which was proposed at the Baltimore meeting, was adopted: After the words "two years" in the fourth line of the second paragraph of Article V there shall be added *and one member elected annually by each division*, making the paragraph read: The Council shall consist of the president, vice president, secretary-treasurer, the retiring president, and the chairman of the Board of Editors of the journal of the Society, with two members elected, one each year, who shall serve for a term of two years, and one member elected annually by each division.

### REPORTS OF COMMITTEES

*The Committee on Ways and Means* made no formal report. It recommended, however, that its duties be delegated to the Advisory Board and the Committee discharged, and on motion this was done.

*The Committee on Standards and Methods* reported through its chairman that it had held two meetings and that tentative recommendations along certain lines would be ready for submission to members of the Society during the year. The Committee hopes that these preliminary recommendations will be carefully considered by the members and that action on them can be taken at the next annual meeting. On motion the report was accepted and the committee continued.

*The Committee on Standard Chart for Percentage Estimates of Injury to Diseased Plants* reported that it finds the chart now in general use by the Office of Cereal Pathology, United States Department of Agriculture, best adapted for most phytopathological purposes. It therefore brings this chart to the attention of the Society and recommends it as worthy of more general use. On motion the report was accepted and the committee discharged.

*The Committee on Continuation of Extension and Investigational Emergency Work of the Department of Agriculture* reported that it visited Washington after the Baltimore meeting and interviewed officials of the Department of Agriculture and Members of Congress, and that it was found impossible to secure continuation of the work desired. On motion the report was accepted and the committee discharged.

### REPORT OF THE ADVISORY BOARD

*The Advisory Board of American Plant Pathologists* reported through its chairman, Dr. G. R. Lyman, on the work of the year. The report showed material progress in all lines of activity assigned to the Board by vote of the Society at the Baltimore meeting.

The Advisory Board has been accepted by the National Research Council as its Committee on Phytopathology, and the Chairman of the Board is serving as a member of the executive committee of the Division of Biology and Agriculture of the Council. During the year a number of important matters were referred by the Council to the Board for advice, and this advisory function of the Board will increase in importance as the work of the Council develops. Through the Board the American Phytopathological Society will be constantly in close touch with the work of the Council and matters of phytopathological interest can be brought immediately to the attention of the Council at any time. The Council has also assisted the Board financially.

A list of the active phytopathological projects in progress in the country for 1919 was prepared and distributed by the Board, and it is felt that a fairly general response was made to this the first request for information of this kind. The list for 1920 will be prepared in cooperation with the Division of Research Information of the Council and should be more complete and valuable than the list for 1919.

During the year three important conferences of workers were held. The first of these was a conference of pathologists, horticulturists, botanists, and growers interested in problems relating to the growing, storage, and marketing of sweet potatoes, and in the diseases of southern crops, such as cotton, corn, and tomatoes. This was called by Prof. H. W. Barre, commissioner for the southern section, and held at Birmingham, Ala., February 26–28, 1919, in connection with the annual meeting of the Association of Southern Agricultural Workers. Forty-six persons, representing thirteen States and the United States Department of Agriculture, were in attendance. After full discussion of all phases of the general sweet-potato problem specific recommendations for future cooperation were adopted, and a committee of three, Prof. H. W. Barre, of Clemson College Agricultural Experiment Station, Prof. G. C. Starcher, Horticulturist at the Alabama Polytechnic Institute, and R. G. Hill, of the Bureau of Markets, United States Department of Agriculture, was appointed to assume leadership in this work. During the year material progress was made and a second conference will be held in connection with the next meeting of the Association of Southern Agricultural Workers in February, 1920.

The second conference was held at Portland, Oregon, April 23 and 24, 1919, under the leadership of Prof. H. P. Barss, Commissioner for the West, on pine-blister rust. This gathering was attended by pathologists, foresters, lumbermen, timber owners, quarantine officials, and others interested in the preservation of the western white-pine industry, and was very important, inasmuch as it was the first occasion on which all those interested in western white pine and its preservation from the blister rust met for consideration of their common problems.

The third meeting was a field meeting held on Long Island June 24–28, 1919, for the consideration of potato diseases, with particular reference to leaf roll and mosaic. The purposes of this conference were to get together as many potato pathologists as possible in a field meeting where informal discussion would be held; to have associated with them in the conference entomologists, agronomists, horticulturists, and others interested in the pathological problems of the potato; to secure international interest through the attendance of foreign plant pathologists; and to promote further coöperative work on potato diseases. About one hundred persons were in attendance. The success of the meeting was due largely to the committee on arrangements, consisting of Prof. M. F. Barrus, and Messrs. W. A. Orton, E. J. Wortley, Paul A. Murphy, and C. R. Orton. Many special plantings and field tests had been arranged in anticipation of the conference.

. Not only was the meeting of great importance in securing a wider knowledge of and interest in certain potato diseases, but the occasion was notable because of the presence of five representatives from foreign countries. Besides Mr. E. J Wortley, Director of Agriculture, Bermuda, and Mr. Paul A Murphy, Pathologist, Dominion of Canada, three distinguished potato pathologists from Europe were in attendance: Dr A D. Cotton, Mycologist of the Board of Agriculture, England; Dr George H Pethybridge, of the Board of Agriculture, Ireland; and Dr. H. M. Quanjer, of the Pathological Laboratory, Wageningen, Holland. The presence of these foreign pathologists, who spent some time in this country following the Long Island meeting, proved a great stimulus to all those who were so fortunate as to meet them The Advisory Board hopes to secure the attendance of other foreign pathologists on the occasion of future meetings.

During the year progress was made on certain cooperative projects, among the most active of which were those on potato seed treatment under the leadership of Dr. I E. Melhus, and on potato spraying, under the leadership of Prof G R. Bisby

Among the other activities of the Advisory Board the most important was the consideration of the proposed Phytopathological Institute. This project was first brought to the attention of the Board by resolutions adopted by the cereal pathologists at St. Louis in June, 1919. The Board has been considering the nature of such an institute and learning the sentiment of American plant pathologists, and has secured the active interest and cooperation of the National Research Council. The matter has now reached the point where it must receive the careful consideration of the American Phytopathological Society. It was discussed at the annual dinner of the phytopathologists.

The Board reports with great regret the resignation of Mr. Paul A. Murphy as commissioner for Canada, due to his approaching departure from America to pursue pathological work in Ireland with Doctor Pethybridge. Mr. Murphy has been a most active and valuable member of the Advisory Board and his loss will be felt by all American pathologists.

### REPORT OF THE TREASURER, 1919

| | | |
|---|---|---|
| *Receipts:* | | |
| Balance from 1918 | $370.04 | |
| Members dues | 2,355.00 | |
| Exchange | 1.52 | |
| Interest | 23.57 | $2,750.13 |
| *Expenditures:* | | |
| Member subscriptions transferred to PHYTOPATHOLOGY | $1,178.00 | |
| Clerical assistance | 135.15 | |
| Postage and supplies | 32.30 | |
| Traveling expenses of secretary to annual meeting | 11.29 | |
| Printing and stationery | 124.79 | |
| Annual appropriation to PHYTOPATHOLOGY | 200.00 | $1,681.53 |
| Balance | | $1,068.60 |
| Balance due sinking fund from 1918 | 315.58 | |
| Amount due sinking fund from 1919 | 445.00 | 760.58 |
| Actual balance | | $308.02 |

During the year a membership drive, organized by the secretary-treasurer, with the coöperation of members of the Society in the various States and in Canada, yielded a large measure of success. Eighty-four persons were nominated during the year for membership and paid their dues for 1919 and their names were placed on the mailing list for PHYTOPATHOLOGY. The formal election of these candidates is necessary at this meeting. In addition sixty others have been proposed for membership beginning with 1920. This makes a total of one hundred and forty-four new members to be elected at this meeting. Sixteen members were suspended this year for non-payment of dues, and there were four deaths and two resignations. Deducting this loss of twenty-two we have a net gain of one hundred and twenty-two, which brings our total membership up to five hundred and twelve. Of this total five are full-paid life sustaining members, eighty-two are life sustaining members paying currently, and four hundred and twenty-five are regular members. Without doubt further large additions can be made to our membership list and each member of the Society is urged to continue his efforts to add new names during the year 1920.

### FINANCIAL STATEMENT OF THE BUSINESS MANAGER OF PHYTOPATHOLOGY, 1919

| | | | |
|---|---|---|---|
| *Receipts:* | | | |
| Balance from 1918 | | $451.99 | |
| Subscriptions and sales | | 1,479.85 | |
| Wortley for illustrations in PHYTOPATHOLOGY | | 15.00 | |
| Appropriation from American Phytopathological Society | | 200.00 | |
| Gift from M. L. Delafield | | 50.00 | |
| Advertising, 1918 | | 193.28 | |
| Member subscriptions | | 1,178.00 | |
| Interest on sinking fund | | 60.00 | |
| Sale of single copy of No. 4, Vol. VIII | | .85 | $3,628 97 |
| *Expenditures:* | | | |
| Manufacture of journal: | | | |
| No. 10, Vol. VIII | $259.16 | | |
| No. 11, Vol. VIII | 159.73 | | |
| No. 12, Vol. VIII | 321.10 | | |
| No. 1, Vol. IX | 314.78 | | |
| No. 2, Vol. IX | 375.91 | | |
| No. 3, Vol. IX | 240.05 | | |
| No. 4, Vol. IX | 193.26 | | |
| No. 5, Vol. IX | 202.45 | | |
| No. 6, Vol. IX | 327.18 | | |
| No. 7, Vol. IX | 221.57 | | |
| No. 8, Vol. IX | 241.31 | $2,856.50 | |
| Miscellaneous bills, Williams & Wilkins Co | | 173.78 | |
| Insurance | | 5.40 | |
| Clerical work | | 41.17 | $3,076.85 |
| Balance | | | $552.12 |

During 1918 a volume of 627 pages was issued. During 1919, notwithstanding an increase of approximately 29 per cent in cost of printing, a volume of 587 pages was issued. We face a still further increase of about 22 per cent in cost of producing PHYTOPATHOLOGY in 1920, which necessitates a further and considerable reduction in the number of pages for Volume X. The rapidly increasing cost of manufacturing the journal must receive the very careful consideration of the Council.

An auditing committee, consisting of C. W. Edgerton and F. D. Fromme, was appointed to examine the accounts of the treasurer and business manager. This committee reported the accounts correct and the reports were adopted.

### RESOLUTIONS

A Committee on Resolutions regarding deceased members was appointed by the president. This committee, consisting of Donald Reddick and E. A. Bessey, offered the following resolution, which was unanimously adopted by a rising vote: "Within the past year there have passed from among us the following members of our Society: Samuel M. Bain, A. T. Bell, William Gilson Farlow, and Kenneth B Laird. The Society assembled in annual convention places on record its sincere regret at the passing of these faithful and earnest colleagues, and with sorrow inscribes on its minutes this expression of its esteem for associates removed from our midst."

### MISCELLANEOUS BUSINESS

The secretary's report of the tenth annual meeting, at Baltimore, as published in No. 4, Volume IX of PHYTOPATHOLOGY, was adopted without correction.

The Society voted to appropriate from any of its available funds the sum of two hundred dollars for the use of PHYTOPATHOLOGY in 1920.

On recommendation of the Council it was voted that two members at large be added to the Advisory Board of American Plant Pathologists, and it was also recommended that the Advisory Board organize with an executive sub-committee to facilitate more frequent meetings for consideration of important business, especially that relating to the activities of the National Research Council.

It was voted unanimously that the American Phytopathological Society endorse most heartily the action of Section G in recommending to the American Association for the Advancement of Science the continued financial support of Botanical Abstracts.

By vote of the Society the secretary was requested to add to future nomination blanks space for the nomination of representatives of the American Phytopathological Society on the Board of Control of Botanical Abstracts.

The consideration of the establishment of a Phytopathological Institute, begun at the Phytopathologists' dinner, was continued by the Society, and, after discussion, the following resolutions, passed at the dinner, were unanimously adopted:

"*Resolved*, That the time has come when the importance of the work of phytopathologists fully justifies the establishment of a Phytopathological Institute—an Institute for the promotion of fundamental research in the broader field of phytopathology and for the stimulation of such research in educational and research institutions and among the members of the phytopathological profession both in this country and abroad.

"*Resolved*, That the American Phytopathological Society authorize the Executive Council, in conjunction with the Advisory Board of this Society, to proceed with full power by the appointment of special committees and by such other procedure as may be necessary to the realization of the establishment of a Phytopathological Institute."

During the discussion following the session devoted to the consideration of the topic Eradication of Plant Diseases the following resolutions were adopted.

"*Resolved*, That the Advisory Board foster vigorously eradication as a means of disease control by bringing together for mutual consideration all interested parties as growers, transportation companies, merchants, consumers, farmers, and public officials

"*Resolved*, That the Society believes that this movement can best be served by a more active and better supported plant disease survey of the United States Department of Agriculture in cooperation with the States."

The serious congestion of the program, due to the large number of papers presented, was discussed. While this condition was recognized as a sign of healthy growth and activity, some measure of relief is necessary. On motion the matter was referred to the Council for action.

It was unanimously voted—

"That the Society heartily approves the efforts of the Advisory Board to promote and correlate cooperative experimental work; that the members of the Society pledge themselves actively and vigorously to prosecute the projects now under way insofar as time and funds permit; that the Society urge that the Board canvass the membership to ascertain which other lines of work should be inaugurated and pursued cooperatively; that it is the sense of the meeting that cooperation on potato seed certification, spraying, and dusting fruits, and other similar problems be organized immediately."

On motion the Chair appointed G. R. Lyman, Donald Reddick, and C. L. Shear a committee to codify the working rules of the Society and to revise the constitution in accordance therewith, the revision to be submitted to the Society for adoption at the next annual meeting.

The following resolution was unanimously adopted:

"The American Phytopathological Society, assembled in annual meeting at St. Louis on January 2, 1920, by unanimous vote desires to extend to the Society of Economic Entomologists and to Mr. E. D. Ball in particular an appreciation of the progress made in the investigation of leaf-hopper injury to potato, and further desires to express regret that through inadvertence in arrangements we as a body failed to hear the discussion of the Entomologists on this subject."

The Secretary was requested to send a telegram of greetings to Dr. Wm. A Orton, president-elect of the Society, who was unable to attend the meeting. He was also requested to convey the appreciation of the Society to the manager of the Marquette Hotel, to the local committee on arrangements, and to the Missouri Botanical Garden and others for the accommodations provided for the Society and the many courtesies extended during the meeting.

### ACTION OF THE COUNCIL

The Council reported the following action for the information of members of the Society:

PHYTOPATHOLOGY. The rapidly increasing cost of manufacturing PHYTOPATHOLOGY presents a problem of grave importance. A careful estimate of the resources of the journal for the year 1920 indicates that it will probably be possible to issue a volume of approximately 450 pages, notwithstanding the increased printing charges. In view of this fact the Council does not think it expedient to take steps at this time to raise membership dues. Moreover there is hope of financial assistance through the Division of Biology and Agriculture of the National Research Council, as this Division is endeavoring to assist biological publications. The interests of Phytopathology will continue to receive careful attention on the part of the Council, with a view to taking such action as future circumstances may render advisable.

Because of the rapidly decreasing stock of back volumes of Phytopathology the price of such volumes was raised from five to six dollars, and the price for single numbers, when such can be sold without breaking a complete volume, was raised to one dollar where there were six issues per volume, and was fixed at 50 cents where there are twelve issues per volume.

*Advisory Board.* The Council selected Prof. A. H. Reginald Buller, of the University of Manitoba, to succeed Mr. Paul A. Murphy as commissioner for Canada. Prof. H. P. Barss and Prof. H. W. Barre were reappointed to succeed themselves as commissioners for the West and the South, respectively. The Council selected Dr. L. R. Jones and Dr. E. M. Freeman as commissioners at large.

*Phytopathological Institute.* In conjunction with the Advisory Board the Council considered further steps looking to the establishment of a Phytopathological Institute. Dr. E. M. Freeman was selected as leader in securing funds. All members of the Society are requested to consider themselves as members of this finance committee and to send Doctor Freeman suggestions as to sources of financial support or regarding the nature and work of such an institute.

*Program of the annual meeting.* In accordance with the vote of the Society the Council considered the problem of relieving the congestion of the annual program. It was not deemed expedient to extend the length of the session beyond four days, nor was it considered advisable at this time to run simultaneous sessions. The secretary of the Society was requested to send out the call for titles and abstracts in the future in time to permit the printing and distribution of the abstracts, together with the program, in advance of the annual meeting. It was suggested that the titles be grouped as far as possible according to the subject matter, one subject or group of subjects being assigned to each session. If the abstracts are prepared with sufficient care it was thought that in the future the entire time of the session can be devoted to discussion. The Advisory Board was requested to assist the secretary in working out the details of the program in accordance with these suggestions.

[Phytopathology for March (10: 119–184) was issued March 12, 1920.]

# PHYTOPATHOLOGY

VOLUME X NUMBER 5

MAY, 1920

# OBSERVATIONS ON SOME COMMON AND IMPORTANT DISEASES OF THE RHODODENDRON[1]

HENRY SCHMITZ

WITH PLATE XI

The purpose of the present paper is to briefly discuss some of the common and more important diseases of the native and cultivated Rhododendrons of the Pacific Coast. It is fully appreciated that some of the diseases here discussed have been described in other connections, and their presence on Rhododendron reported, but their relations to this host seem to have been little investigated. It must be understood that the treatment which follows does not attempt to be exhaustive in detail, but the work was undertaken with the idea of bringing together in one place the important diseases of this shrub on the Pacific Coast.

## ARTIFICIAL INOCULATIONS

The general texture of the Rhododendron leaf is very tough, so that great difficulty was at first experienced in keeping the spores moist long enough to insure germination and the proper establishment of the mycelium. To overcome this difficulty, glass rings were sealed to the leaf with a mixture of one part of paraffin and three parts of beeswax. The spores in water were then placed within and a cover glass sealed onto the ring. This kept the spores under excellent conditions for germination.

With some fungi, such as Sporocybe, spores were formed in artificial culture only after long periods of time, while in other instances, as Cryptostictis, the mycelium failed to produce spores under the various conditions of temperature maintained, even after a year's growth. This made it necessary in some of the artificial inoculation experiments to take the spores directly from the bud or leaf as the case might be. This method is not in strict accord with common practice in artificial inoculation experiments, but there is sufficient evidence that the causal agent was the particular fungus in question.

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[1] The writer wishes to express his indebtedness to Dr. J. W. Hotson of the University of Washington, under whom the work was done, for his kind suggestions and helpful criticisms; to Prof. Roland Thaxter for assistance in the determination of some of the fungi; and to the Missouri Botanical Garden for the privileges of the herbarium.

In all cases cultures were again made from the artificially inoculated area and the fungus therein reobtained and compared with the original fungus.

### SPOROCYBE AZALEAE (PK.) SACC.

*Sporocybe azaleae* produces a "bud rot" disease of Rhododendron. Plants affected by the disease are easily recognized at all times of the year, the brown, dead buds being very conspicuous in contrast with the whorl of green leaves which surrounds them. Examination of the diseased bud shows the fruiting bodies of the fungus protruding in all directions.

In the early stages of infection, the bud scales become only slightly browned; later they become dark brown, and still later the fruiting bodies appear. The fungus may also work down the stem, killing the bark and the cambium. Plate XI, figures 6 and 10, illustrate the appearance of this disease on the buds, branches, and pods.

Buds artificially inoculated with spores of *Sporocybe azaleae* in May produced fruiting bodies of the fungus by September. Spores of the fungus were shaken down between the bud scales with little or no injury to the bud. The buds so infected did not open but were killed by the fungus, while buds slightly injured in a similar manner and not inoculated opened normally.

Bud rot is perhaps the most serious disease of the native *Rhododendron californicum*, and is yearly becoming more prevalent on the cultivated varieties. Spraying will probably not serve to control it on the cultivated varieties, owing to the deep-seated nature of the disease in the buds, and it is suggested that the buds be cut out and burned as they become infected.

### MELAMPSOROPSIS PIPERIANA ARTHUR

*Melampsoropsis piperiana*, or "Rhododendron rust," is found in great abundance on the leaves of *Rhododendron californicum*. It has not been found by the writer on the cultivated varieties.

Melampsoropsis produces numerous small, reddish-yellow pustules on the lower surface of the leaves. These may at times be so numerous that they run together, forming a large blotch. Very often the margin is the only portion of the leaf affected, forming a reddish-yellow line completely around the leaf, about one-half centimeter from the edge. The effects of the rust are quite conspicuous, and the disease is easily distinguished from the other diseases of the Rhododendron by the yellowish spores.

Two large spore crops seem to be produced each season in this region. During the latter part of April (1915) a great number of leaves were found to be affected and spores were very abundant. By September of the same year only an occasional spore-bearing leaf was found. Late in November, however, great numbers of spores were found in the same locality. This periodic formation of spores may, of course, be entirely dependent upon weather conditions.

As has been said, the rust was not found on Rhododendrons under cultivation, but there is no reason why it might not occur on a cultivated plant. If it should occur, spraying with Bordeaux mixture in April would no doubt be helpful.

Plate XI, figure 1, shows the characteristic appearance of leaves affected with *Melampsoropsis piperiana*.

### WITCHES' BROOMS

Conspicuous brooms are common on the native Rhododendron. They may reach considerable size and attain great age.

The exact cause of the broom has not been determined. Cultures made from portions of the branches of the brooms in numerous cases produced a mycelium very much resembling that of *Sporocybe azaleae*. The mycelium did not produce spores and therefore could not be identified.

Whether *Sporocybe azaleae*, *Melampsoropsis piperiana*, or some other fungus or insect causes these brooms is a point to be cleared up by future investigations.

The general appearance of the brooms is shown in plate XI, figure 3.

### WHITE LEAF

By "white leaf" a peculiar leaf condition is referred to in which a leaf or cluster of leaves may be partially or entirely devoid of chlorophyll. This condition is quite common in the case of *Rhododendron californicum*. There is no evidence of any fungus or other injury of any kind on such leaves.

Very often, although not always, this condition is found in connection with "witches' brooms." The trouble may be non-parasitic or it may be due to the action of a fungus lower down on the stem or in the roots. If the latter condition is the cause, it is somewhat analogous to the "white spot" condition occurring on alfalfa.

### LOPHODERMIUM RHODODENDRI CES.

*Lophodermium rhododendri* Ces. is reported as causing a leaf disease of Rhododendron both in Europe and in the United States. It is common on *Rhododendron californicum* on the Pacific Coast.

The fungus produces small, boat-shaped perithecia on the upper surface of the leaves. The deadened portions of the leaves usually have an irregular marking encircling from three to seven of the fruiting bodies. This condition is shown in plate XI, figure 4.

As a rule, the fungus is found on leaves entirely dead and lying on the ground. Whether the fungus originally killed the leaves or whether the defoliation was due to other causes, is not known. In several cases living leaves with only small dead areas were found. *Lophodermium rhododendri* was the only fungus observed on the dead areas. This would suggest that the fungus mentioned may be the parasite involved.

Owing to the imperfect condition of the material, no pure cultures of Lophodermium were obtained.

### COCOMYCES DENTATUS (KEZ. ET SCHM.) SACC.

The exact species of Coccomyces here involved is questionable, but is provisonally referred to *Coccomyces dentatus* (Kez, et Schm.) Sácc. It is very abundant both on the leaves and twigs of the native Rhododendron.

The effects of the fungus on the leaf are similar in appearance to those produced by *Lophodermium rhododendri*, except that in this case the perithecia are more or less star-shaped.

Fruiting bodies were never found except on dead leaves, and it is still a question whether or not Coccomyces is the killing agent. The fungus is, however, found on living stems and seems eventually to kill the twigs.

Very good material of the fungus was not found. Although numerous attempts were made, no pure cultures were obtained and satisfactory inoculation experiments were therefore precluded. The general appearance of leaves and twigs affected by this fungus is shown in plate XI, figures 2 and 9.

### CORYNEUM RHODODENDRI S.

This species of Coryneum was found quite prevalent on the leaves of cultivated varieties of Rhododendron. The fungus causes irregular dead areas on the leaves. Very often a dead strip entirely around the leaf may be found. There is also some evidence that it grows on the portions of the leaves killed by aphids. Leaves slightly injured with a hot needle and inoculated with spores of the fungus produced the typical disease and spores after two and one-half months' growth under greenhouse conditions. The controls showed no further changes than the small deadened areas due to the hot needle punctures.

Plate XI, figure 8, shows the general appearance of leaves infected with *Coryneum rhododendri.*

### SPHAERELLA RHODODENDRI CKE.

This fungus is found very commonly on the leaves of both the native and cultivated varieties of Rhododendron.

The dead areas have a deep reddish-brown to brown color. The upper surface is slightly roughened, due to the presence of numerous minute perithecia. Very often the dead area includes the tip half of the leaf (plate XI, fig. 5).

No cultures were obtained from the leaves that could be definitely identified as Sphaerella, hence artificial inoculations were made by placing small portions of infected leaves in contact with slightly injured leaves, but in no case did infection result.

In the opinion of the writer, the species of Sphaerella here in question is not parasitic on Rhododendron, but seems to appear on the leaves only after they have been partly or entirely killed by summer or winter desiccation. In its natural habitat, the Rhododendron often suffers from lack of water, and dead areas appear on the leaf which may in every way be identical to those on which Sphaerella is found, except that the fungus is lacking.

### PESTALOZZIA GUEPINI DESM.

The Pestalozzia disease of Rhododendron was found only on plants under cultivation. The deadened areas of the leaves affected by the fungus are usually small and are more or less gray in color. The fungus is often found on leaves after they have suffered from the activities of a small aphid that is quite common in the early spring. The young leaves are affected. Small deadened areas appear, which later enlarge because of infection by Pestalozzia. Plate XI, figure 7, shows the general appearance of the leaves after they have been attacked by the aphid and in the various stages of secondary infection with Pestalozzia.

The usual methods of artificial inoculation were employed and positive evidence was obtained showing that this species of Pestalozzia is parasitic in nature.

### CRYPTOSTICTIS SP.

By this name I refer to a disease found commonly both on the native and cultivated forms of Rhododendron. The area of the leaf killed by this fungus is usually circular or partly so in shape. The concentric rows of fruiting bodies produced by the fungus are plainly visible, and it is by means of these concentric rings of spore pustules that this disease may be easily distinguished from the other leaf diseases here discussed.

The spores have somewhat the shape and general appearance of spores of Coryneum, and may be mistaken for that form in a hasty observation. They have, however, a characteristic appendage.

In artificial cultures at first a dense, downy mycelium was formed, which later shrank to quite limited dimensions. Spores were not produced on the types of nutrient media used nor under the conditions of growth maintained. For this reason, the spores for the artificial inoculations were taken directly from a diseased leaf. These inoculations showed the fungus to be parasitic in nature, and concentric rings of spore pustules were produced some three months after the inoculations were made. The controls showed no further change than the small deadened areas due to the punctures of the hot needle.

LABORATORY OF FOREST PATHOLOGY,
SCHOOL OF FORESTRY
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## PLATE XI

FIG. 1. *Melampsoropsis piperiana* Arthur. Characteristic appearance of infected leaves.

FIG. 2. *Coccomyces dentatus* (Kez. et Schm.) Sacc. The dark, star-shaped perithecia are common both on the leaves and twigs.

FIG. 3. Witches' broom. A comparatively small dense broom.

FIG. 4. *Lophodermium rhododendri* Ces. Numerous fruiting bodies of the fungus are evident on the upper surface of the leaf.

FIG. 5. *Sphaerella rhododendri* Cke. It is quite probable that Sphaerella rhododendri is saprophytic on the dead areas of the leaves.

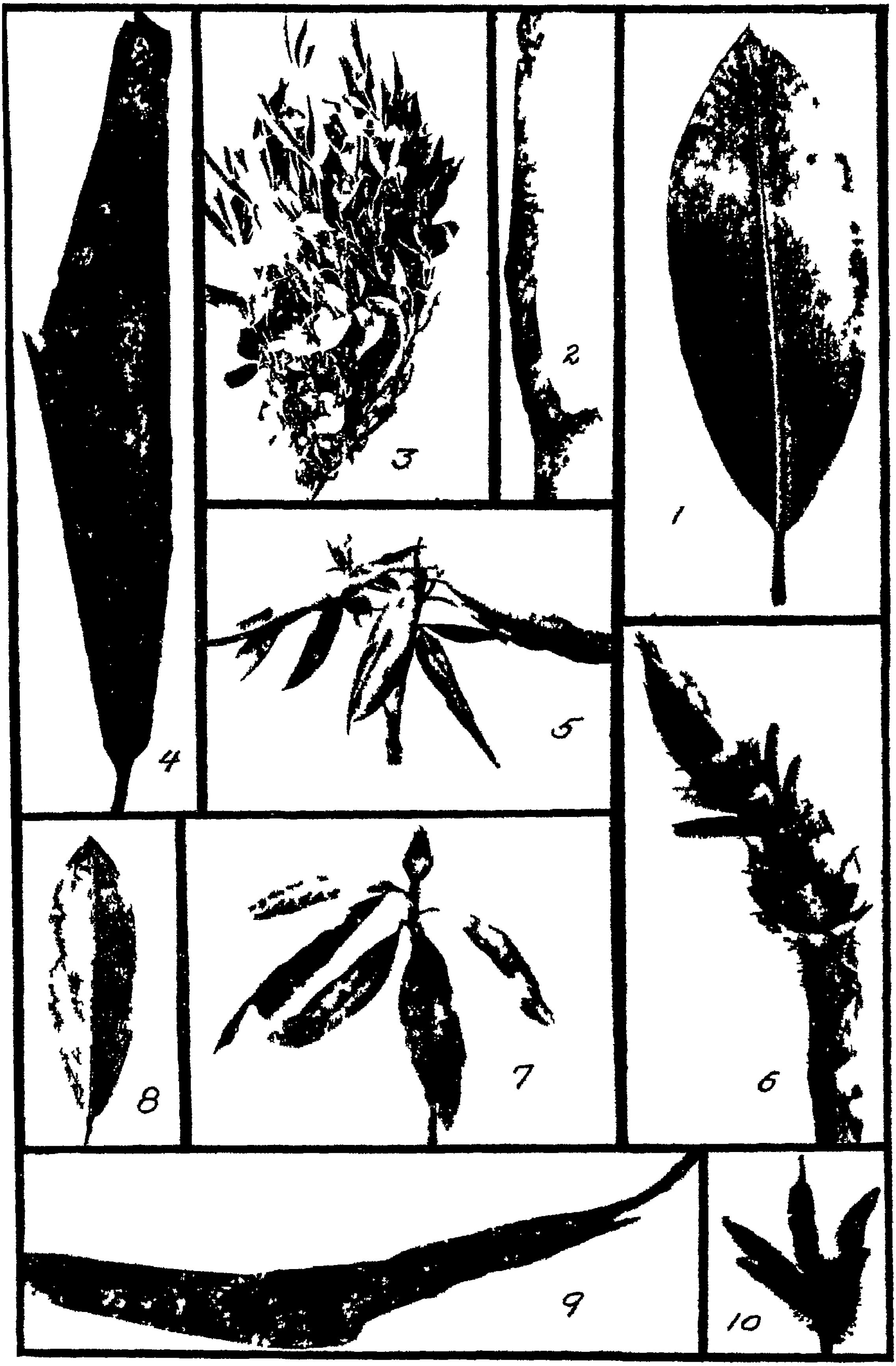
FIG. 6. *Sporocybe azaleae* (Pk.) Sacc. The fungus kills the branches as well as the buds. Branch infection usually results from bud infection as here shown.

FIG. 7. *Pestalozzia guepini.* Desm. A typical Pestalozzia infection. The infected areas may, however, increase in size until the entire leaf is killed.

FIG. 8. *Coryneum rhododendri* S. The infection is usually more or less confined to the periphery of the leaf.

FIG. 9. *Coccomyces dentatus* (Kez. et Schm.) Sacc. The fruiting bodies appear as small spots on the light-colored areas of the leaf.

FIG. 10. *Sporocybe azaleae* (Pk.) Sacc. An infected seed pod.



SCHMITZ RHODODENDRON DISEASES

# FACULTATIVE HETEROECISM IN PERIDERMIUM CEREBRUM AND PERIDERMIUM HARKNESSII

E. P. Meinecke

With Two Figures in the Text

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In the year 1916 report[1] was made of successful infections of *Pinus radiata* as a result of direct inoculation with aeciospores of *Peridermium harknessii* Moore from *Pinus radiata.* Owing to totally inadequate laboratory and greenhouse conditions, the experiments at that time had been confined to two plants with two control plants. One inoculated plant died; on the other, seven inoculations were made, of which five produced typical galls and aecia.

It was concluded that, if *Peridermium harknessii* Moore was indentical with *Peridermium cerebrum* Peck [*Cronatrium cerebrum* (Peck) Hedgc. and Long], field observations as well as inoculation results made it appear that in California, at least within the range of the old *Peridermium harknessii,* the aecial form on one side and the uredinial and telial on the other must be highly independent of each other, and that the heteroecism of the fungus is facultative.

The revolutionary character of the result of the inoculations made it imperative that the experiments be repeated on a larger scale, in order to put the facts themselves beyond the reach of criticism, before an interpretation of the phenomenon in the light of prevailing theories could be attempted.

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[1] Meinecke, E. P. Peridermium harknessii and Cronartium quercuum. Phytopathology, 6: 225–240, 2 fig. Je. 1916.

At the time of publication all endeavors to connect the so-called *Peridermium harknessii* with *Peridermium cerebrum* had failed. Since then, Hedgcock[2] has succeeded in producing uredinia and telia on *Quercus velutina* with aeciospores from *Pinus radiata*, thereby indicating the identity of the coast form with *Peridermium cerebrum* Peck. Still it must be noted that to the present day no pycnia have been found on the thousands of the coast galls examined, and that the many inoculation experiments with aeciospores on *Quercus agrifolia* and *Q. californica*, as well as with teliospores on *Pinus radiata*, have failed to produce results. *Quercus agrifolia* is very commonly associated with *Pinus radiata* and often harbors the uredinia and telia of what is called *Cronartium quercuum*. While the failure to infect the alternate host may possibly be due to a series of unfavorable circumstances, the absence of pycnia is distinctly surprising. Neither do they appear in nature, nor can they be coaxed into development *in vitro*.

The old *Peridermium harknessii* Moore included not only the coast form, now identified with *Peridermium cerebrum*, on *Pinus radiata* and other pines, but also a gall-producing Peridermium on mountain pines, *Pinus sabiniana*, *P. ponderosa*, *P. jeffreyi*, and *P. contorta*. *Pinus radiata* occurs in abundance on the coast at Monterey. *Pinus sabiniana* inhabits the lower fringe of dry foothills, while *Pinus ponderosa* sets in from about 1500 feet and extends to about 6000 feet elevation. *Pinus jeffreyi* becomes abundant from about 5000 feet on, reaches its optimum at 6000 to 6500 feet, and ranges to over 9000 feet. *Pinus contorta* in the central Sierra Nevada grows at an altitude of 6000 to 10,000 feet.

The first mention of *Peridermium harknessii* Moore is made by H. W. Harkness in 1880[3] as follows: "*Peridermium harknessii* n. sp. Moore. On *Pinus ponderosa*, Colfax." Together with this we find: "*Peridermium pini* Fr. On *Pinus insignis*. San Francisco." In 1880 *Peridermium pini*, a European form on *Pinus silvestris*, was still considered to embrace a stem-inhabiting form (*Peridermium pini corticola*) and a needle-inhabiting form (*Peridermium pini acicola*). It may be assumed that Harkness, finding a Peridermium on the stems and *Peridermium californicum* on the needles, associated on *Pinus radiata* (*P. insignis*), followed the error of contemporaneous European authors and considered both as *Peridermium pini*.

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[2] Rhoads, A. S., Hedgcock, G. G., Bethel, Ellsworth, and Hartley, Carl. Host relationships of the North American rusts, other than Gymnosporangiums, which attack conifers. Phytopathology, 8: 309–352. Jy. 1918. Literature cited, p. 346–352.

[3] Harkness, H. W., and Moore, Justin P. Catalogue of the Pacific Coast fungi. Read before the California Academy of Sciences, Feb. 2, 1880. p. 27. [San Francisco, 1880.]

Four years later Harkness[4] dropped *Peridermium pini* and retained only *Peridermium harknessii.* He writes (p. 37): "*Peridermium harknessii* Moore. Spermogones unknown. Protospores: Peridia aggregate, irregular, encircling the stem; spores irregular, very finely echinulate, orange, becoming white. μ. 35–40. On stems and branches of *Pinus insignis, P. ponderosa* and *P. sabiniana.* Appearing as annular swellings, often two feet in circumference upon the trunks and branches. In the Sierra Nevada, below the height of 4000 feet and in the Coast Range. Particularly abundant at Monterey. The different species of Peridermium of the pine seem to be doubtfully distinct. H. W. H."

Harkness' type specimens in the California Academy of Sciences were partly rescued from destruction in the conflagration of San Francisco in 1906. We find under no. 2698 (subnumber 449) the bottom of a small pill box with the subnumber 449 (1). The box evidently contained the specimen of which no traces are left. A note in what appears to be Harkness' handwriting reads: "Peridermium found on pine trees near the town of Colfax April 26, '76,—on *Pinus ponderosa* on hillside in rear of the Station. *Peridermium harknessii.*" Colfax lies in the foothills on the California side of the Sierra Nevada at an elevation of 2400 feet. The missing specimen must then be considered as the type specimen, collected on *Pinus ponderosa* at Colfax, Cal.

While the type specimen itself has disappeared, there exists a duplicate in the Ellis Collection of the New York Botanical Garden. This specimen is very well preserved and consists of a piece of a branch lengthwise cut in two and glued onto a label with the following legend in Dr. Ellis' handwriting: "*Peridermium harknessii* Moore on *Pinus ponderosa*, Colfax, Calif. Dr. H. W. Harkness (28)." The specimen shows two distinct galls, a larger and a smaller one, which cannot be distinguished from the common form on the pines of the Sierrra Nevada. There cannot be any doubt that Dr. Ellis received this specimen from Harkness and that Harkness himself considered it typical for *Peridermium harknessii* Moore and that the specimen was collected at the type locality.

It is clear that Harkness was not familiar with the gall form on *Pinus contorta* or *Pinus jeffreyi,* both of which occur above 4000 feet elevation, and that his *Peridermium harknessii* (Moore never published on the species) originally referred to the gall form on pines on the lower slopes of the Sierra Nevada. It was only later that he extended this designation to the gall form on *Pinus radiata.*

The galls on the coast pines and those on the Sierra pines are, at first glance, so much alike that they have long been regarded as identical, all the more so since the only Cronartium associated with the High Sierra form had already been connected with *Peridermium filamentosum.* We know now[5] that this Cronartium in reality represents two different forms, one going to the gall form, the other to *Peridermium filamentosum* (syn. *P. stalactiforme* Arth. and Kern), both on *Pinus contorta, P. ponderosa,* and *P. jeffreyi.* The latter Peridermium is the aecial stage of *Cronartium filamentosum* (Peck) Hedgc. and Long. There appears no reason why the former should not be called *Cronartium harknessii* nov. comb.

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[4] Harkness, H. W. New species of Californian fungi. Bul. California Acad. Sci., No. 1: 29–47. 1884.

There is still a great deal of confusion with regard to these last two forms, owing to the fact that both produce apparently identical uredinia and telia on scrophulariaceous hosts. Weir and Hubert[6] in the same paper report experiments with "*Cronartium coleosporioides* (D. and H.) Arth. (*P. stalactiforme* type)," with "*Cronartium coleosporioides* (typical gall form)," and with "*Cronartium coleosporioides* (typical blister type)." Presumably the "typical gall form" refers to Cronartium sp.? of Rhoads, Hedgcock, Bethel, and Hartley,[7] that is, *Cronartium harknessii.* The "*Peridermium stalactiforme* type" is undoubtedly *Peridermium filamentosum* (Peck) Hedgc. and Long. It is less easy to place the "typical blister type," unless here also the *P. stalactiforme* type (*P. filamentosum*) is meant.

At the present time *Peridermium filamentosum* and *P. stalactiforme* are considered identical. Certain striking morphological differences may have a deeper meaning. On *Pinus contorta* the infection of the stem often takes place through small twigs and spreads very evenly up and down the trunk much faster than sideways. The result is a very neat elongated diamond. This form of development does not seem to occur on *Pinus ponderosa* or *P. jeffreyi,* at least not in the Pacific Coast mountains. The aecia on *Pinus ponderosa* often appear in the shape of very conspicuous long cylinders with an unusually tough peridium. This form seems to be unknown on *Pinus contorta.* It is not impossible that the present term *Peridermium filamentosum* covers two different species, both of which go to Castilleia and related forms and there produce uredinia and telia which are indistinguishable from each other and from those of *P. harknessii.* In that case the Castilleia group would harbor

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Rhoads, A. S., Hedgcock, G. G., Bethel, Ellsworth, and Hartley, Carl. Op. cit., p. 317.

Weir, J. R., and Hubert, E. E. A serious disease in forest nurseries caused by Peridermium filamentosum. Jour. Agr. Research, 5: 781–785. Ja. 24, 1916.

These results have recently been confirmed by the author of this paper.

[6] Weir, J. R., and Hubert, E. E. Recent cultures of forest tree rusts. Phytopathology, 7: 106–109. Ap. 1917.

[7] Op. cit., p. 317.

three uredinia and telia which can only be separated by cultural studies. Experiments are on the way to clear up this point.

We have, then, two gall-forming Peridermia on pines on the Pacific Coast: one erroneously called *Peridermium harknessii* Moore, now *Peridermium cerebrum* Peck (fig. 1), on *Pinus radiata*, *P. attenuata*, and *P. muricata*, and another on *Pinus sabiniana*, *P. ponderosa*, *P. jeffreyi*, *P. contorta*, and perhaps *P. attenuata*, representing the true *Peridermium*

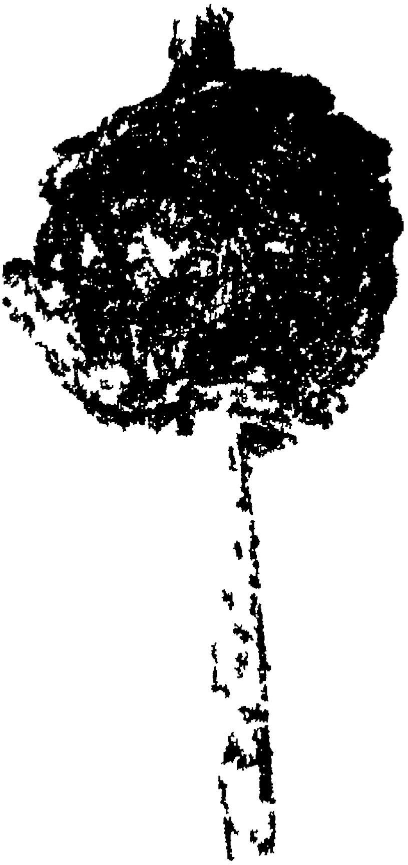

FIG. 1

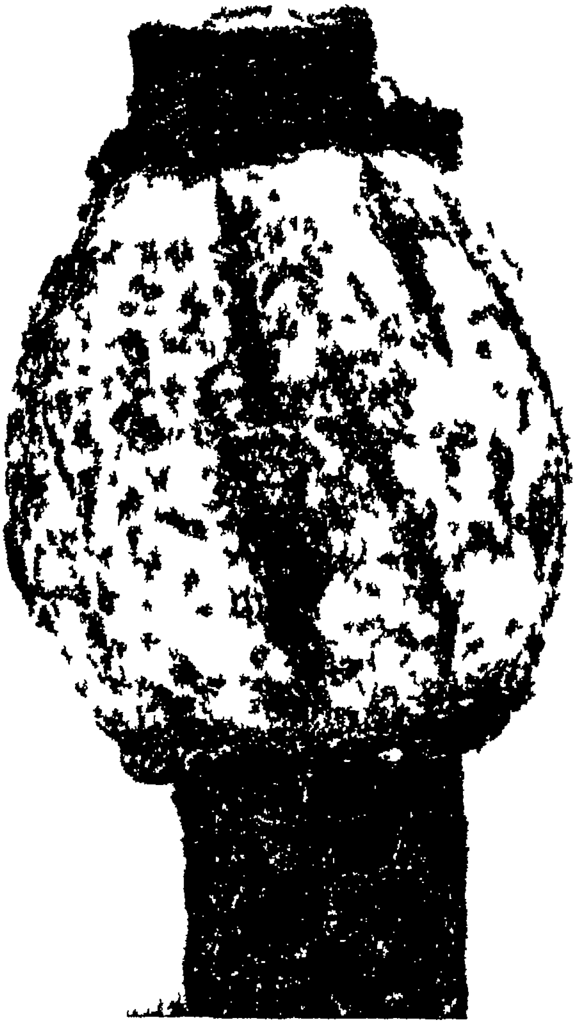

FIG. 2

FIG. 1. MATURE GALL OF PERIDERMIUM CEREBRUM PECK ON PINUS RADIATA

The aecia break forth in the cracks between the bark scales. (Natural size.)

FIG. 2. MATURE GALL OF PERIDERMIUM HARKNESSII MOORE ON PINUS PONDEROSA

Note the collar and the large, naked, spore covered surfaces resulting from the peeling off of large pathological bark scales. (Natural size.)

*harknessii* Moore (fig. 2). The latter form is still thought by Weir and Hubert[8] to be identical with *Peridermium filamentosum* (Peck) Hedgc. and Long. The same authors[9] consider the various caulicolous forms of rusts occurring on *Pinus contorta* and *P. ponderosa* in the Rocky Mountain region as the aecial stages of *Cronartium coleosporioides*, and believe it "not only possible but probable that the same forms on *Pinus contorta* and *P. ponderosa* as known elsewhere in the western United States belong here also." Rhoads, Hedgcock, Bethel, and Hunt[10] are undoubtedly right in refusing to accept this view. There are vast regions in the Sierra Nevada where *Pinus contorta, P. ponderosa*, or *P. jeffreyi* are affected either with the gall-forming rust or with the form producing no or inconspicuous swellings, to the absolute exclusion of the other. If both were identical, surely one would expect to find the forms intermingled.

In the early stages the galls of *Peridermium cerebrum* as found on *Pinus radiata* and other coast pines are difficult to distinguish from those of *Peridermium harknessii* on *Pinus contorta, P. jeffreyi, P. ponderosa*, and *P. sabiniana*. Later, even before the appearance of the aecia, the galls of *P. harknessii* become rough, and the bark breaks open, the cracks showing plates of young, corky tissue of a chestnut-brown color, while the galls of *P. cerebrum* generally remain smooth for a while, even after the formation of aecia has begun. The aecia of the latter are comparatively small, and appear singly and irregularly scattered, later becoming more or less confluent. Relatively small pathological bark scales keep pace with the growth of the gall and the development of the aecia. The aecia of *P. harknessii*, on the other hand, are much larger and become confluent to such an extent that they often form very large spore-producing surfaces. The pathological bark scales are large and come off easily, leaving wide, smooth spore-covered areas exposed. These pathological bark scales finally reach to the cambium and make further formation of aecia on those areas impossible. The scales break away and the smooth, naked, hypertrophied wood of the gall appears. Another characteristic of the *P. harknessii* gall is the formation of a collar of dead bark standing out from the upper and lower end of the gall. This collar as a rule is absent in *P. cerebrum*, as are also in general the large bark scales and the wide areas of exposed surfaces. The peridium of *P. cerebrum* is rather delicate and ruptures irregularly, while the circumscissile peridium of *P. harknessii* often persists in the form of broader flakes. In general, the galls of *Peridermium cerebrum* are sphaeroid to pear-shaped in their youth and later become more and more spherical, while those of *Peridermium harknessii* show less of a tendency to become truly spherical but remain more pear-shaped, well set off below against the branch of the host and tapering to the upper end. There are, however, many exceptions to this rule.

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[8] Op. cit., 1916, p. 783.

[9] Op. cit., 1917, p. 107.

[10] Op. cit., p. 317.

About 30 to 50 per cent of the galls both of *Peridermium cerebrum* and of *P. harknessii* are accompanied by witches'-brooms.

On the basis of the descriptions given above it is possible to distinguish mature galls of *Peridermium cerebrum* on the coast pines and of *P. harknessii* on the mountain pines.

Harkness' specimen in the Ellis Collection of the New York Botanical Garden answers in every detail the description given here for *Peridermium harknessii.* The collar is very pronounced in both galls, sharply setting off above and below the large and smooth spore-producing surfaces. In the rich collections of the San Francisco laboratory of Forest Pathology, both forms could be segregated without much difficulty. All galls of *Pinus contorta, P. jeffreyi, P. ponderosa,* and *P. sabiniana* were typically of the *Peridermium harknessii* type. All mature galls on *Pinus radiata* showed the characters of *P. cerebrum.* Of five specimens of galls on *Pinus attenuata,* however, three plainly belonged to the *P. cerebrum* type while two approached the *P. harknessii* type. *Pinus attenuata* has a range extending from the ocean shore to over 5000 feet in the Sierra Nevada, that is, from the range of *Peridermium cerebrum* well into the range of *Peridermium harknessii.* It may well be that this pine is susceptible to both forms. The presence of uredinia (*Uredo coleosporioides* Diet. and Holw., so called) on *Castilleja foliolosa* and others near the coast may be explained as coming from infections from *Peridermium harknessii* on *Pinus attenuata,* though the mild climate of the California Coast makes overwintering in the uredinial form on Castilleja quite probable. The telial form has not yet been found.

Inoculations of *Castilleja foliolosa* from San Francisco with aeciospores of *Peridermium cerebrum* from *Pinus radiata* failed.

Since the publication of the first paper in 1916, *Peridermium cerebrum* has been found on *Pinus muricata* at Santa Barbara, Cal., and on a cultivated specimen of *P. halepensis* at San Mateo, Cal. Uredinia as well as telia were collected in August and September on *Quercus californica*; uredinia at Gilroy, Santa Clara Co., Cal., telia at the same place, both by J. W. Sheldon, and uredinia near Sonora, Cal., by the author. At Gilroy the nearest pines were at least five miles away.

Unfortunately a series of experiments on *Pinus radiata* with pedigreed material (results of the former inoculations) was lost when the inoculated plants died owing to poor greenhouse facilities. The new series have not yet shown results.

The experiments here reported and inaugurated for the purpose of confirming the first success were made with wild material.

The same method of inoculation was employed throughout. The aeciospore material was dusted or shaken onto a small quantity of tap water in a watch crystal. It should not be taken with an instrument directly from the aecium, in order to avoid a possible admixture of viable mycelium. With a small sharp scalpel tangential slits about 0.5 to 0.75 cm. in length and not more than 0.5 cm. in width were cut in the living bark of the test plants. The scalpel, after effecting a longitudinal incision through the cortex, passes the cambium without cutting the wood and makes its exit through the epidermis opposite to the line of entrance so that the slits extend through all cortical tissues. The resulting membrane of cortical tissues remains attached above and below, but can easily be bulged out to allow insertion of the inoculum. The latter is introduced with the same scalpel and the membrane is pressed into position. The lips of the wound readily stick together and the membrane fully protects the inoculum from drying out. No further precautions are necessary beyond the usual sterilization of tools, etc., and the protection of the plants from spurious infection, either with bell jars or with celluloid tubes as described by Hubert, until the wounds are healed.

In the earlier experiments the wounded part of the stem was wrapped with cotton which was kept moist for a number of days and then removed. It appears unnecessary to follow this procedure.

All the material used came from typical, more or less spherical, hemispherical to pyriform galls, in which the aecia form separately, are sometimes confluent, but hardly can be called typically cerebroid.

*Inoculations on Pinus radiata with aeciospores from Pinus radiata.* Including the first reported experiment, thirty-eight inoculations on fifteen plants were made under controlled conditions. Of these, nineteen have been successful. Some of the galls resulting are richly sporulating, others are still too young to fruit. All are typical in shape and all developed from the inoculated wound. All control plants are perfectly sound.

*Inoculations on Pinus muricata with aeciospores from Pinus radiata.* Eleven plants were inoculated in twenty-two places. Of these inoculations, twelve were successful, typical galls being formed. One of these has two aecia at the present day.

*Inoculations on Pinus radiata with aeciospores from Pinus attenuata.* One plant was inoculated in four places. One of these four inoculations was successful. The gall produced is sporulating richly.

The galls invariably developed at the point of inoculation and at this point only. All check plants remained sound.

In the foregoing experiments more plants were inoculated than are here given. From different causes a number of the young inoculated trees died. Some of these had developed small galls. They are, however, not counted as positive results.

Accepting only definite results, the total number of infections of *Pinus radiata* and *P. muricata* with aeciospores from *Pinus radiata* and *P. attenuata* amounts to thirty-two out of a total of sixty-four inoculations, or 50 per cent.

Analyzing the effect of the time of inoculation on the percentage of results, we arrive at the following table:

| HOSTS | DATE OF INOCULATION | WOUNDS INOCULATED | POSITIVE INFECTION | PER CENT |
|---|---|---|---|---|
| P. att.-P. rad. | Mar. 26 | 4 | 1 | 25 |
| P. rad.-P. rad. | Apr. 14 | 4 | 1 | 25 |
| P rad -P. rad. | Apr. 14 | 3 |  | 0 |
| P. rad.-P. mur. | Apr. 18 | 6 |  | 0 |
| P. rad.-P. rad. | Apr. 18 | 10 | 3 | 33 |
| P. rad.-P. rad. | May 10 | 14 | 10 | 71 |
| P. rad.-P. mur. | May 10 | 16 | 12 | 75 |
| P. rad.-P. rad. | May 23 | 7 | 5 | 71 |

While inoculations earlier than May are not impossible, the best chances for positive results seem to lie in the month of May, due either to the then attained maturity of the spores or to the condition of the inoculated host. In nature the galls begin to fruit as early as December and are usually in full sporulation in the months of March and April. It is to be noted that for a large part of the inoculations here reported, plants of the same origin were used. The plants of *Pinus radiata* and *P. muricata* inoculated on April 18 and May 10 respectively were all raised together from seed in the nursery. Here the result stands 0 and 33 per cent as against 71 and 75 per cent.

During the most favorable time the average of positive results amounted to 63 per cent.

In no case were pycnia or anything resembling these bodies noticed. This tallies with observations of thousands of galls in nature. The microscope so far has failed to reveal any indication of abortive or internal pycnia. Pycnia are common in *Peridermium cerebrum* of the East. In

Japan they are produced in such frequency[11] that "children lick the abundant spermogones on account of the sweet juice that oozes from them."

The periods after which swelling of the stem and finally sporulation set in are variable. The time of the year at which the experiments were carried out had no influence on the incubation period, since all inoculations were made within two months of each other, the extreme dates being March 26 and May 23. The distance at which the plants were kept from the laboratory, on account of poor housing facilities, did not permit regular inspections. In one inoculation from *Pinus radiata* onto *P. radiata* the first swelling was observed after eleven months, while sporulation did not begin until thirty-five months after inoculation. In the experiment on the same pines reported in 1916 the first swellings were noted after seven months and sporulation began twenty-one months after inoculation. The corresponding figures for inoculation of *Pinus radiata* from *P. attenuata* are sixteen and twenty-nine months, of *Pinus muricata* from *P. radiata* eight and eleven and one-half months, of *Pinus radiata* from *P. radiata* (new series) eight and eleven months. The figures are given simply as general indications, since the plants have not been under constant observation and the periods elapsed between the different stages may be too long by a month or two. Differences in the growing conditions under which the plants were kept may account in a measure for the variations reported.

Gall formation was accompanied by typical witches'-brooms in eleven out of thirty-three cases. In some of the more recent inoculations witches'-brooms may still develop.

The galls produced are very typical of *Peridermium cerebrum* on *Pinus radiata.* They have the characteristic shape. In their youth they are either spherical or hemispherical, or they are more or less fusiform to pear-shaped, the gall being rather well set off against the sound part of the stem. Later the fusiform gall develops into the more characteristic spherical shape. In the younger galls the bark is smooth, showing plainly the scar of the inoculation wound. Later the bark becomes roughened, and after prolonged sporulation the dead bark scales give the gall surface a rugged appearance. The aecia appear as well defined, separate sori, which later may become partly confluent. The peridium is rather delicate and evanescent. The aecia break forth between the bark scales and continue to develop from year to year until the gall dies off. On older galls during the maximum of sporulation large parts may be covered with the orange colored spore masses, but the aecia are always easily traced.

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[11] Grove, W. B. The British Rust Fungi. p. 24. Cambridge, 1913.

The spores produced are in all respects morphologically identical with those described for *Peridermium cerebrum*. Their measurements agree fully with those of the latter.

Galls were formed on parts of the stem from one to four years old. An inoculation of a five-year-old internode remained without result.

If there could exist a doubt as to the validity of the infections published in 1916, the results given here establish as a definite, incontrovertible fact that it is possible to infect young plants of *Pinus radiata* with aeciospores of *Peridermium cerebrum* from *Pinus radiata* and *P. attenuata*, and plants of *Pinus muricata* with spores from *Pinus radiata*, and that the resulting more or less spherical galls are in all details identical with the galls so common on various coast pines of California. In other words, the aeciospores of a heteroecious rust, capable of producing uredinia and telia on oaks, are also capable of producing aeciospores on pines by omitting the uredinial and telial stage altogether.

## FURTHER INOCULATION EXPERIMENTS

Aeciospores from the result of the direct aecial infection of *Pinus radiata* reported in 1916 were used repeatedly for inoculations on young plants of *Quercus agrifolia* and on *Q. engelmanni* (March 1, March 21, March 25, and April 2). More plants of *Quercus agrifolia, Q. chrysolepis, Q. wislizeni*, and *Q. engelmanni* were inoculated with aeciospores from *Pinus radiata* of various collections (dates of inoculation Feb. 13, April 2, April 15, May 10, and May 22). Inoculations cannot be made later than early spring because the oak leaves are in general fully matured in May. The experiments extended over several years. Without exception the inoculations failed to produce results.

The following inoculations were made with uredinial or telial material from oaks:

*March 9.* Urediniospores from *Quercus agrifolia* on *Quercus lobata.*

*March 29.* Urediniospores from *Quercus agrifolia* on *Q. lobata.*

*April 2.* Teliospores from *Quercus californica* on *Pinus radiata* (introduction of telial horns with well developed sporidia in wounds).

*April 18.* Teliospores from *Quercus agrifolia* on *Pinus radiata* (same method).

*August 26.* Teliospores from *Quercus californica* on *Pinus radiata* (same method).

All of these inoculations gave negative results. They were carried on through several years. The plants were kept under most favorable growing conditions.

Hedgcock[12] reports successful inoculations of leaves of *Quercus rubra*, *Q. emoryi*, *Q. marilandica*, *Q. gambelii*, and *Q. lobata* with urediniospores taken from the leaves of *Quercus gambelii*. *Quercus lobata* is an oak common in California, within the region in which *Peridermium cerebrum* is indigenous.

Besides these laboratory and greenhouse experiments, a considerable number of inoculations were made in the open, with due precautions, on leaves of sound *Quercus agrifolia* with aeciospores of *Peridermium cerebrum* on *Pinus radiata*. Not in a single case did they result in infection, although at the same time and shortly after, uredinial sori appeared on other oaks in quantity. It must be borne in mind that in nature many oaks seem to be perfectly immune, while others standing in the closest proximity to them are more or less heavily infected. The fact points to the existence of immune strains in *Quercus agrifolia*. If this is true, laboratory experiments will always be uncertain until it is possible to raise young oak plants from acorns of a tree known to be susceptible to infection. Unfortunately, the uredinial form is least frequently found on trees large enough to bear acorns.

While this difficulty may explain the failure to get positive results from inoculations on oaks, it does not do away with the corresponding failure to infect young pines with sporidia from oaks. Further inoculations of both types are on the way. It is not easy to secure good telial material. Telia are not very common. Their small size adds to the difficulties of collection. The period during which they mature appears to be rather short. It seems to vary from year to year and does not always coincide with the most favorable stage of development of the oak plants on hand.

### EXPERIMENTS WITH PERIDERMIUM HARKNESSII

*Peridermium cerebrum* is not the only rust fungus with facultative heteroecism. A series of inoculation experiments with *Peridermium harknessii* has shown that direct transfer of the fungus from pine to pine is equally possible.

The aeciospores of *P. harknessii* very readily produce uredinia and telia on *Castilleja miniata*. Inoculations on *Castilleja foliolosa* failed, perhaps due to the poor condition of the plants, which are difficult to raise in the laboratory.

In inoculating pines with aeciospores of *Peridermium harknessii* the same "slit" method was used as in the experiments with *Peridermium*

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[12] Hedgcock, G. G. Notes on some diseases of trees in our national forests. I. 4 p. Washington, D. C., 1914. Paper read at the meeting of the American Phytopathological Society at Boston, Mass., December 30, 1909.

*cerebrum*. The galls are in full sporulation in July. The following is a synopsis of the results obtained.

| HOSTS | DATE OF INOCULATION | WOUNDS INOCULATED | POSITIVE INFECTION | PER CENT |
|---|---|---|---|---|
| P. pond.-P. rad. | July 21 | 3 | 1 | 33 |
| P. cont.-P. cont. | July 25 | 13 | 12 | 93 |
| P. cont.-P. jeffr. | July 25 | 21 | 12 | 57 |
| P. jeffr.-P. jeffr. | July 24 | 16 | 10 | 62 |
| P. jeffr.-P. cont. | July 24 | 13 | 10 | 77 |

Of a total of sixty-six inoculations forty-five, or 68 per cent were successful. In each case a gall was found exactly at the place of inoculation and at no other place. All control plants remained sound. The forty-five galls are accompanied by witches'-brooms in seventeen cases or 37 per cent.

In one inoculation, made in 1915, gall formation took place after eighteen months, while sporulation did not set in until forty-four months after inoculation. The remaining experiments are still too recent to show aecia. The galls appeared after eleven and twelve months. In no case could pycnia be observed. The transfer from *Pinus ponderosa* to *P. radiata*, the typical host of *Peridermium cerebrum* on the Coast, is of particular interest.

The results prove that the aeciospores of *Peridermium harknessii* are equally capable of producing uredinia and telia on certain Scrophulariaceae and aecia and aeciospores on pines.

## KLEBAHN'S EXPERIMENTS

Two years after the author's publication of the results of direct aecial transmittal from pine to pine, Klebahn[13] published similar results with *Peridermium pini* in support of Haack's[14] inconclusive experiments. The rigorous blockade before the entrance of the United States into the war evidently kept American scientific periodicals from reaching Germany, and Klebahn consequently was not aware that the problem of direct aecial infection had already been solved. The blister rust of *Pinus silvestris* in Europe consists, according to Klebahn, of two forms, one connected with *Cronartium asclepiadeum* (Willd.) Fries, which Klebahn calls *Peridermium cornui* Rostr. and Kleb., and another morphologically almost identical one, but not connecting with any Cronartium, called by him *Peridermium pini* (Willd.) Kleb. It is with the latter that Klebahn experimented. He operated with young trees raised from the seed of infected trees. The inoculations were made partly with wounds, partly without wounds. In both cases he obtained positive results, though more infections occurred through wounds. About 30 per cent of his inoculations were successful. The resulting aeciospores germinated readily on a film of water. The type of germination was invariably aecidioid, without a trace of promycelia and sporidia. It is particularly noteworthy that some of the infections resulted in the formation of pycnia. At the time of Klebahn's report these particular infections had not yet produced aecia. Klebahn is disinclined to believe that heteroecism exists in *Peridermium pini* side by side with the repetition of aecia. "Perhaps the acquisition of the faculty to produce aeciospores from aeciospores is accompanied in this case by the loss of heteroecism and the disappearance of teliospores." He proposes to solve this question indirectly through similar experiments with the undoubtedly heteroecious *Peridermium cornui*, and suggests experimentation with *Peridermium strobi*.

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[13] Klebahn, Heinrich. Peridermium pini (Willd.) Kleb. und seine Übertragung von Kiefer zu Kiefer. Flora, 111–112: 194–207, ills., pl. 4–5. 1918.

[14] Meinecke, E. P. M., Op. cit., p. 237.

## GERMINATION STUDIES

Fromme[15] in a criticism of the author's first paper suggests that germination studies and perhaps cytological studies may be necessary in order to establish the true character of the spores, referring to the aeciospores used in the experiments. Since the publication of the first paper the author has made a great number of germination tests with the aeciospores resulting from infection with aeciospores of *Peridermium cerebrum* and *P. harknessii* as well as with spores from wild galls. The spores were dusted on a drop either of 5 per cent gelatine or, in by far the greater majority of cases, on a drop of tap water on slides which were placed on moistened filter paper in petri dishes. Germination under such conditions is always very abundant. In every case, without any exception, the single germ-tube produced was typically aecidioid. The tube growing into the air reaches great length, ten to fifteen times the length of the spore, is delicate and tapering towards the tip, occasionally branched, and often spirally curved, but it never shows the slightest tendency to produce sterigmata or sporidia, or any character which could possibly be interpreted as belonging to a promycelium. In order to leave no doubt on this point a very large number of spores (roughly about 800,000) were examined. No difference could be observed in the behavior of the spores used for inoculation and the spores resulting from the infection.

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[15] Fromme, F. D. Facultative heteroecism (?) of Peridermium harknessii and Cronartium quercus. Phytopathology, 6: 411–412. 1916.

After two to three days the long germ-tubes are generally overgrown by moulds. In order to hold back the latter as well as to stunt the germ-tubes and induce them to develop typical promycelia on shorter tubes in case they were capable of doing so, a number of spore cultures were exposed to low temperatures. The result was a general elimination of the moulds and a very decided stunting of the germ-tubes, but no promycelia or anything resembling them were obtained.

It was thought possible that the actual contact with the tissues of the host plant might be necessary to stimulate the spores to produce promycelia and sporidia. A number of inoculations by the slit method were made and the spores examined after one to six days. In every case was the germ-tube typically aecidioid. Nothing even remotely resembling a promycelium or basidiospores could be detected.

## FACULTATIVE HETEROECISM

Fromme[16] has taken exception to the use of the term facultative heteroecism as applied to the faculty of omitting the alternate host, perhaps correctly so from his point of view, which considers *Peridermium harknessii* Moore in the older acceptance of the name and *Peridermium cerebrum* Peck as distinct. To anyone who believes that the California coast form connects with *Cronartium quercuum*, the term should appear logical. Hitherto heteroecism has been considered as obligate where it occurred in rust fungi, including *Peridermium cerebrum* Peck. Hedgcock's successful cross infection proves that the fungus can still exercise its faculty of heteroecism, while the direct infections from pine to pine show that it can omit the oak phase. Its heteroecism is no longer obligate, but facultative, optional. If a doubt can still exist concerning the California form of *Peridermium cerebrum*, none is possible with regard to the heteroecism of *Peridermium harknessii*. In other words, the results of our experiments show that heteroecism is not as fixed and rigid a phenomenon as has hitherto been believed.

The mere overwintering of the uredinial stage of *Cronartium cerebrum* does not constitute facultative heteroecism. It is the formation of teliospores and basidiospores which counts. In obligate heteroecism the organism should return to oak. To judge from the failure to infect California oaks the heteroecism of the fungus seems to have lost some of its obligate character.

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[16] Op. cit., p. 411.

Why, according to Fromme, "a species must be capable of producing both generations on the host of one generation to be considered facultatively heteroecious" is not clear. The etymology of the Greek word heteroecism indicates plainly that it means the faculty of different generations of an organism to inhabit different hosts, just as autoecism indicates that an organism inhabits one host or host group only. An organism cannot be facultatively autoecious. By the mere fact that it produces different generations on different hosts it automatically becomes heteroecious by definition. Only heteroecism can be facultative or optional. In autoecism there can be no choice of hosts.

## INTERPRETATION

The outstanding features of our investigations may be summarized as follows:

In California a Peridermium, described by Moore as *P. harknessii* and now referred to *P. cerebrum*, abundantly produces galls on a number of pines, particularly *Pinus radiata*. The galls resemble very much, but not in all detail, the *Peridermium cerebrum* Peck of the East. Pycnia, common in typical *P. cerebrum*, have never been found. Witches'-brooms, rare in *P. cerebrum* of the East, are common on the California coast.

Side by side with *Peridermium cerebrum* there exists in California a sporophytic generation resembling in all respects *Cronartium cerebrum* (Peck) Hedgc. and Long. It occurs on a number of oaks commonly associated with the pines known to be hosts to *Peridermium cerebrum*. Both uredinial and telial stages are found. Infected oaks and pines may be located close together or may be so far distant from each other that inoculation through the air is excluded. Heavily infected individual oaks may stand in closest proximity to perfectly sound pines and vice versa. If both forms belong together, as is now accepted, they certainly show a remarkable independence of each other.

It is possible to infect pines directly with spores from the galls of the same species or certain other species. The percentage of successful inoculations is high. All inoculations of pines with sporidia from Californian oaks or of oaks with aeciospores from pines have failed.

The spores produced in the pine galls resulting from direct infection with spores from pine galls invariably show typical aecidioid germination, as do the spores from all wild pine galls.

The mycelium overwinters on the evergreen oaks and produces urediniospores and teliospores in spring.

On mountain pines a gall form, in gross appearance resembling that of the coast pines, is common, particularly on *Pinus contorta.* The aeciospores of this form, now called *Peridermium harknessii*, readily produce uredinia and telia on *Castilleia sp.* and typical galls with aecia and aeciospores on pines. Pycnia have never been found, with exception of abortive ones in a single case on *Pinus jeffreyi*, which may prove to refer to another Peridermium. Witches'-brooms are common. The resulting aeciospores show true aecidioid germination.

What is the interpretation of these facts?

Fromme[17] is of the opinion that the spores "although morphologically aecidiospores, (they) may be aeciospores, teliospores, or urediniospores in function, since all three may be borne catenulately in aecidioid fructifications. If the inoculated spores are aeciospores, the resultant spores must be urediniospores or teliospores (aeciospores do not reproduce aeciospores)." It is true that no definitely established case has been reported so far in which the aeciospores of a heteroecious rust reproduce aeciospores. Eriksson's results with *Puccinia arrhenateri* (Kleb.) Erikss. have never been confirmed. But what then is an aeciospore? A spore with all the morphological characters of an aeciospore, with aecidioid germination, failing to produce a promycelium, failing to produce sporidia, is difficult to classify as a teliospore. Cytological studies will show whether our spores are binucleate or uninucleate. If the former, they may be either aeciospores or urediniospores. If they are to be regarded as urediniospores we would have, in the same organism, two totally different kinds of urediniospores on two different hosts. Perhaps the spores are uninucleate. That would then be the only remaining character which they have in common with the teliospore, a teliospore in which the basidium itself, without formation of sporidia, directly infects the host. In that case the fungus would present the unique spectacle of forming no aeciospores but two distinct telial forms, also on two different hosts, one resembling in all detail except nuclear behavior true aeciospores, the other acting like normal teliospores. It is evidently too early to accept so extraordinary a possibility.

It seems far more probable that the accepted rule that aeciospores never produce aeciospores does not invariably hold true. Klebahn also considers the resulting spores as aeciospores. Perhaps the omission of the pycnia in the cycle has some significance in connection with the direct transmission of the rust from pine to pine by aeciospores. One might be inclined to assume that the disappearance of the pycnia adds to the sexual vigor of the aeciospore to such an extent as to make the latter sufficiently virulent to attack pines without having to pass through the sporophytic generation on oak or Castilleia respectively. This suggestion would be at variance with Olive's[18] views. He explains the jump from one host to hosts of very different systematic position on the ground of the profound and invigorating stimulus imparted by the sexual fusions. "The diploid aecidiospores, which are produced shortly following these fusions, are endowed with more vigor than any of the rest of the forms of rust spores; and are thus the most capable of infecting foreign protoplasm." Granted that the stimulus imparted by the sexual fusions may explain the jump from one host to another. But what is far more puzzling is that a spore form recently rendered sufficiently vigorous to effect that jump, should not be vigorous enough to re-infect the protoplasm it has so long been accustomed to. It may be, after all, that the omission of so fixed a character as the formation of telia in heteroecious rusts is far more momentous than the change from one host group to another. In *Cronartium harknessii* the percentage of successful inoculations of pines is decidedly lower than the percentage of infections of Castilleia.

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[17] Op. cit., p 412.

In *Peridermium cerebrum* and *P. harknessii* the aeciospores possess both faculties, that of the jump to foreign protoplasm and that of re-infecting the old host. Perhaps such cases are more common than appears on the surface. Is what we observe in these forms an early stage in the development from autoecism to heteroecism, or are we watching the breaking down of heteroecism and an emancipation of generations?

## AECIAL INFECTION AND DISSEMINATION

It is evident that a heteroecious rust possessing, in addition to the usual mode of spreading through basidiospores, the faculty of directly infecting pines through aeciospores is particularly favored with regard to dissemination. This double faculty explains the extraordinary frequency of galls of *Peridermium harknessii* and *P. cerebrum*, both of which are capable of doing considerable damage. Seedlings are often killed. Older trees are stunted or deformed by the large galls on the stems. While certain hosts, such as *Pinus attenuata* and *P. sabiniana,* do not possess commercial value, the loss in *Pinus radiata,* one of the commonest garden and park trees of the coast, or in *P. ponderosa* and *P. jeffreyi,* both important timber trees, is not to be underestimated.

In the light of the experiments reported, it is far from impossible or even improbable that *Cronartium ribicola* may also be transmitted directly from pine to pine. Considering the very great importance of this disease it is highly desirable that similar experiments be carried out with aeciospores of the white pine blister rust.

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[18] Olive, E. W. Origin of heteroecism in the rusts. Phytopathology, 1: 139–149. 1911.

## SUMMARY

To the host list of *Peridermium cerebrum* (the former *Peridermium harknessii* Moore) in California should be added *Pinus muricata* (and *P. halepensis*). The host list of *Cronartium cerebrum* Peck in California now also includes *Quercus californica.*

Previously reported direct infections of *Pinus radiata* with aeciospores from galls of *Peridermium cerebrum* on *Pinus radiata* are confirmed. Other direct infections with *Peridermium cerebrum* spores have been successful from *Pinus radiata* to *P. muricata,* and from *P. attenuata* to *Pinus radiata.*

*Peridermium harknessii* Moore, now confined to the gall form on mountain pines, though retaining its faculty easily to infect scrophulariaceous hosts and there to produce uredinia and telia, is also capable of reproducing galls, aecia, and aeciospores in direct inoculations from *Pinus contorta* to *P. contorta,* from *P. contorta* to *P. jeffreyi,* from *P. jeffreyi* to *P. jeffreyi,* from *P. jeffreyi* to *P. contorta,* and from *P. ponderosa* to *P. radiata.*

The germination of the resulting aeciospores in moist air is invariably truly aecidioid. No promycelium and no sporidia are formed. No pycnia are found. Witches'-brooms frequently accompany the galls.

The heteroecism of *Peridermium cerebrum* and *Peridermium harknessii* is facultative.

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# ON THE AMOUNT OF COPPER REQUIRED FOR THE CONTROL OF PHYTOPHTHORA INFESTANS ON POTATOES

O. BUTLER

WITH THREE FIGURES IN THE TEXT

The year 1919 was a very favorable one for the development of late blight in New England, and therefore also a year eminently suited to a study of the relation between degree of control obtained and total quantity of copper applied per acre. It is of extreme importance that we know the minimum amount of copper required to give control and also the number of sprayings that must be applied with a Bordeaux mixture of any given strength, in order to secure the desired control; in other words, to determine whether frequency of application has any appreciable effect on the amount of copper that must be applied per acre. Upon these points an experiment carried out by the writer during the summer of 1919 supplies data that is not without interest.

In the experiment referred to, a potato field a trifle over 3 acres in extent was divided into twenty-three plots of four rows each, which were then sprayed, that is, all but no. 16, which was left as a witness, in the manner indicated, and with the fungicides named in table 1.

The spraying machine used in the experiments was an Iron Age equipped with triple action pump, and the pressure employed was 180 pounds. At the first spraying one nozzle was used per row, giving an application of 71.5 gallons per acre, while in the subsequent sprayings three nozzles were employed, giving an application of about 135 gallons per acre. When the three nozzles were first used a boom of the type illustrated in figure 1, *b*, was employed but was subsequently changed to the form illustrated in figure 1, *a*, which proved more satisfactory and covered the plants better.

The potatoes were sprayed on July 11 and subsequently thereafter as called for in the schedule (see table 1), the weather never once hindering the work. While the experimental field was located some seventeen odd miles from Durham, the nearest weather bureau station, the climatic data for the latter place will portray sufficiently accurately for our purpose the essential characteristics of the growing season as they pertain to the former. The graph (fig. 2) gives the daily maximum and minimum temperature and the rainfall for the period covered by the experiment.

TABLE 1

*Fungicides used and spraying schedule followed in experiments on control of light blight*

| FUNGICIDE USED | PLANTS SPRAYED WEEKLY | | PLANTS SPRAYED EVERY TWO WEEKS | | PLANTS SPRAYED EVERY THREE WEEKS | |
|---|---|---|---|---|---|---|
| | Plot number | Date sprayed | Plot number | Date sprayed | Plot number | Date sprayed |
| 1 per cent Bordeaux mixture 1 1 | 1–17 | July 11, 18, 24<br>August 1, 8, 16, 22, 29<br>September 6, 12 | 6–20 | July 11, 24<br>August 8, 22<br>September 6 | 11 | July 11<br>August 1, 22<br>September 12 |
| 1 per cent Bordeaux mixture 1 05 | 2–18 | As above | 7–21 | As above | 12 | As above |
| 2 per cent Bordeaux mixture 1 05 | 3–19 | As above | 8–22 | As above | 13 | As above |
| Pyrox | 4 | As above† | 9 | As above | 14 | As above |
| Bordeaux mixture paste* | 5 | As above‡ | 10–23 | As above§ | 15 | As above |

* Manufactured by General Chemical Company, New York

† On August 16, 2 per cent Bordeaux mixture was accidently used instead of Pyrox

‡ Bordeaux mixture paste not used on August 29 and September 6, as our supply of material became exhausted

§ Bordeaux mixture paste applied September 12 instead of September 6 (see note (‡) above)

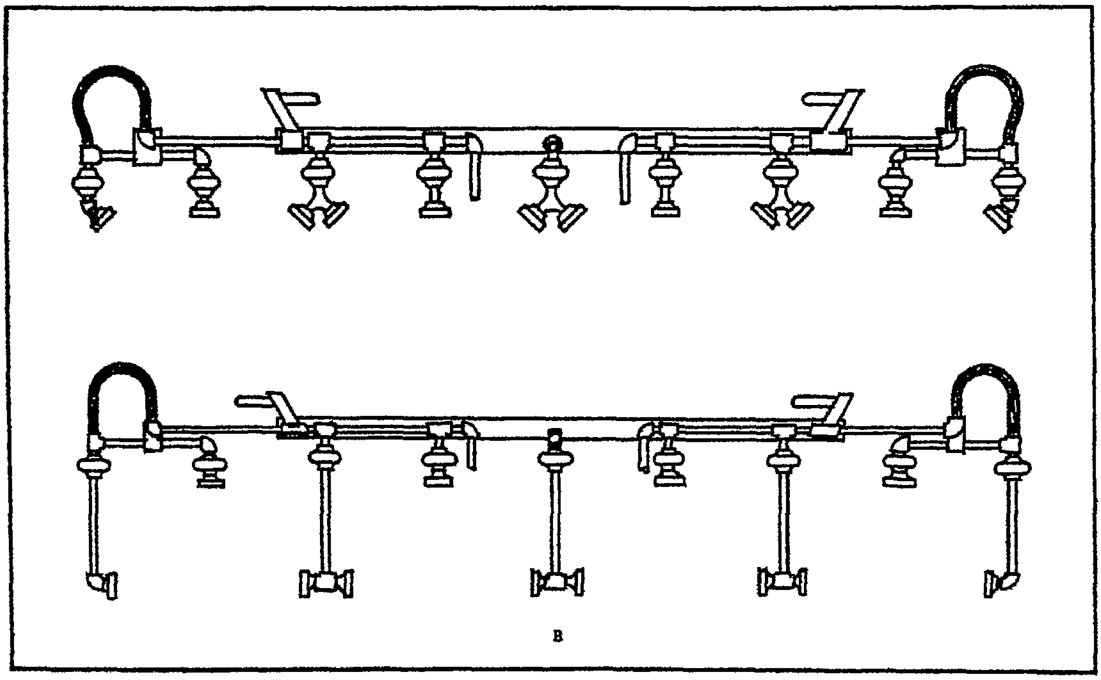

FIG 1 TYPES OF BOOM USED IN SPRAYING EXPERIMENT

It will be noticed that, beginning September 9, we have cool wet weather eminently favorable for the development and spread of late blight, and, in effect, the malady proved extremely violent and destructive throughout the state of New Hampshire towards the close of the growing season. During the wet weather in September the late blight obtained a footing in the experimental field. On September 26 the foliage of the unsprayed plants was dead but the stalks were still green, while one week later the plants were entirely dead. The foliage of the plants sprayed once in three weeks with Pyrox was in bad condition on September 18, and on September 26 had almost completely withered. The foliage of the plants sprayed every three weeks with Bordeaux mixture paste was in poor condition on September 18, but the plants had not completely withered when a severe frost on October 9 completely killed the plants throughout the experimental field. The foliage of the plants in the plots other than those just considered remained green until the end, and showed no *inter se* differences worthy of comment. The foliage injury and premature death of the witness plants and those sprayed with Pyrox every three weeks, as well as the hardly less severe injury noted on the plants sprayed with

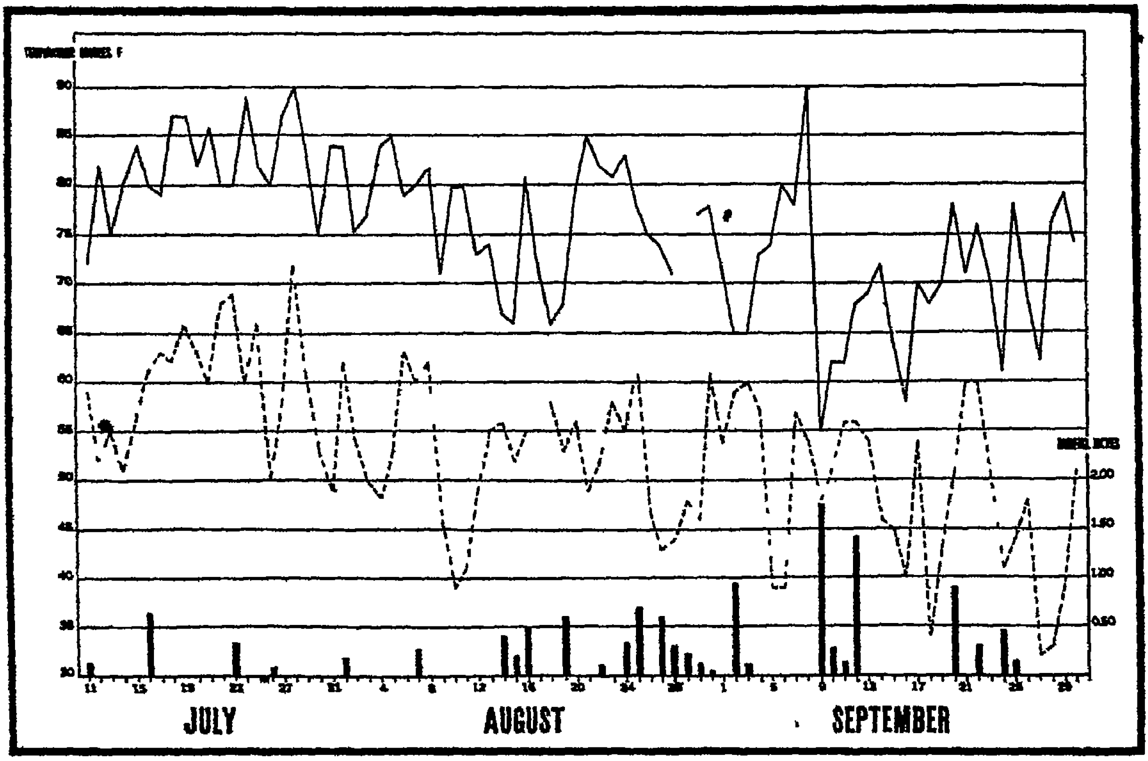

FIG. 2. DAILY MAXIMUM AND MINIMUM TEMPERATURES, AND RAINFALL AT DURHAM, N. H., FROM JULY 11 TO SEPTEMBER 31, 1919

———— = Maximum temperature
– – – – – – – = Minimum temperature
Vertical block column, = Rainfall

Bordeaux mixture paste every three weeks, was in large part due to early blight (*Macrosporium solani*) and not conspicuously to late blight. The late-blight fungus did not find as favorable a *nidus* for growth as would have obtained in the absence of early blight, though it was sufficiently virulent, as the data obtained at harvest will show, to make the experiment conclusive.

During the twelve days beginning October 18 the potatoes were harvested, the entire two central rows of each plot being taken for the data on tuber infection. The results obtained are indicated in table 2, in which is also given the amount of copper applied per acre during the season.

TABLE 2

*Effect on the control of late blight of strength of the fungicide used and of number of applications given*

| FUNGICIDE USED | PLANTS SPRAYED WEEKLY | | PLANTS SPRAYED EVERY TWO WEEKS | | PLANTS SPRAYED EVERY THREE WEEKS | |
|---|---|---|---|---|---|---|
| | Amount copper applied per acre | Per cent late-blight | Amount copper applied per acre | Per cent late-blight | Amount copper applied per acre | Per cent late-blight |
| | *pounds* | | *pounds* | | *pounds* | |
| 1 per cent Bordeaux mixture 1:1.... | 26.04 | 0.35 | 12.38 | 1.90 | 9.65 | 3.62 |
| 1 per cent Bordeaux mixture 1:05... | 26.04 | 0.13 | 12.38 | 0.60 | 9.65 | 3.07 |
| 2 per cent Bordeaux mxiture 1:05... | 52.09 | 0.00 | 24.77 | 0.00 | 19.30 | 0.76 |
| 2.5 per cent Pyrox.................. | 8.91 | 0.64* | 1.83 | 4.65 | 1.43 | 14.28 |
| 2.75 per cent Bordeaux mixture paste. | 21.47 | 1.16† | 12.92 | 1.00† | 10.07 | 3.21 |

* Two per cent Bordeaux mixture 1:05 was applied once by mistake; the figure is therefore too low.

† Owing to lack of material, the spraying schedule was not fully carried out; the figure given is, therefore, too high.

A study of table 2 shows that absolute protection is afforded when between 24.7 pounds and 26 pounds of copper are applied per acre, and that whether this amount is applied in weekly or fortnightly sprayings the results obtained are substantially the same. To apply the necessary amount of copper is more important even than timeliness of application. It will be remembered that weather particularly favorable to the late-blight fungus supervened on September 9, and that conditions favorable to its spread and development continued until the plants were killed by frost. Now if we turn to the spraying schedule we will find that the last three-weekly spraying given before weather favorable to the late-blight fungus set in was on August 22, while the last weekly and fortnightly applications were given on September 6. In other words, in the case of the three-weekly sprayings, weather conducive to the development of late blight supervened eighteen days after the last application had been made, whereas in the case of the weekly and fortnightly applications the interval was only three days. The plants sprayed every three weeks with 2 per cent Bordeaux mixture 1.05 yielded 0.76 per cent diseased tubers, substantially the same amount as 1 per cent Bordeaux mixture 1:05 applied fortnightly; 2 per cent Bordeaux mixture applied fortnightly

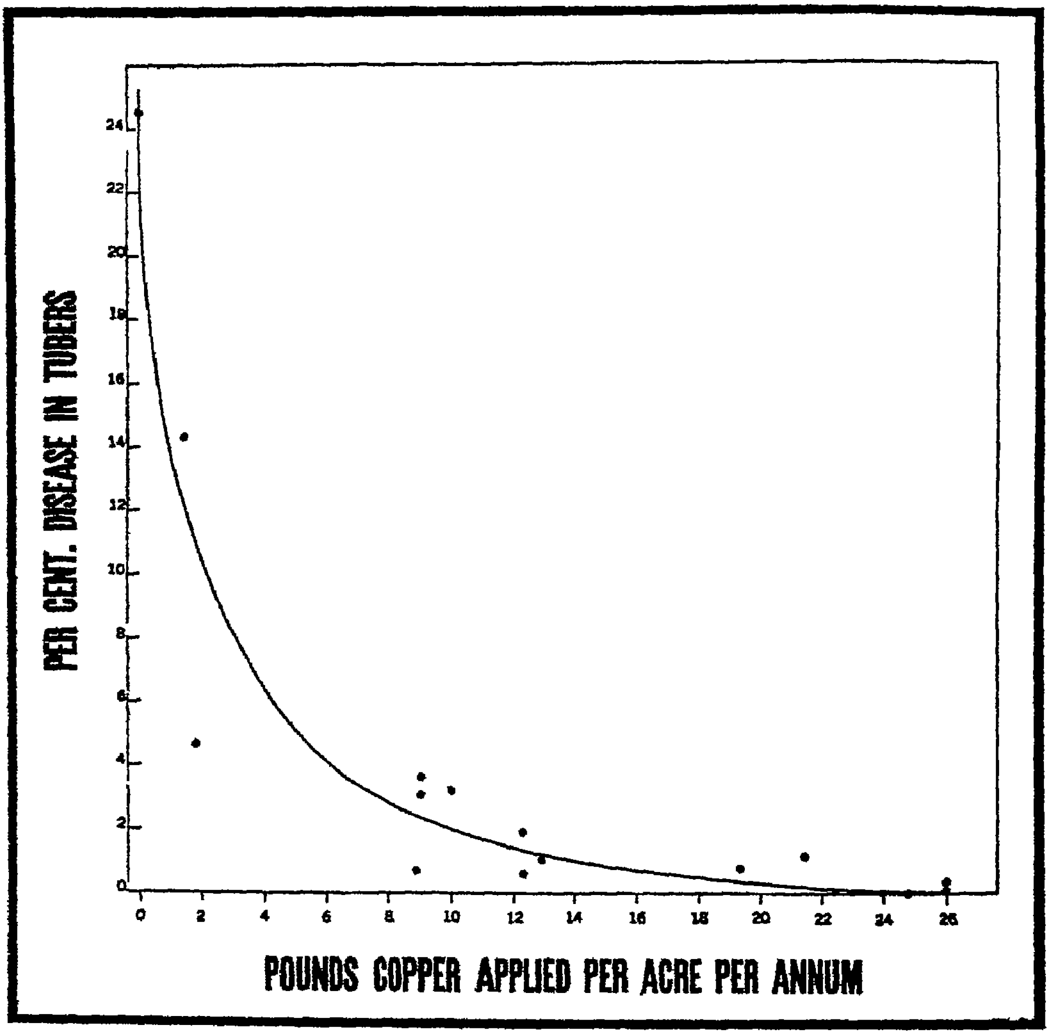

FIG. 3. CURVE SHOWING RELATION BETWEEN PER ACRE ANNUM APPLICATION OF COPPER AND LATE-BLIGHT INFECTION OF POTATO TUBERS

gave absolute protection, while 1 per cent Bordeaux mixture 1:05 sprayed weekly permitted very slight infection. Timeliness of application did not therefore determine the degree of freedom from late blight. If one plots the results secured in the experiment he will find the relation between protection obtained and the amount of copper applied per acre per annum to give the curve shown in the following graph (fig. 3). A study of this curve permits one to deduce that if a 0.5 per cent Bordeaux mixture were used, then the potatoes would have to be sprayed twenty times in sixty-three days, *i.e.*, once every three days, in order to obtain the protection afforded by a 1 per cent Bordeaux mixture 1:05 applied weekly, or a 2 per cent Bordeaux mixture 1:05 used fortnightly. On the other hand, lengthening the interval between sprayings, which would be theoretically allowable, is subject to limitations, as is also the equally permissible increasing of the strength of the Bordeaux mixture employed. In the first place a Bordeaux mixture stronger than 2 per cent becomes difficult to handle; as a matter of fact, even a 2 per cent mixture does not handle satisfactorily, except when made directly in the spray tank, and an interval of more than three weeks between applications of a fungicide would leave new growth unduly long exposed to the dangers of infection. We are therefore obliged to adopt a middle ground. Just what this middle ground shall be must depend on the degree of protection sought and the economy of application desired. It is clear that one can spray with 2 per cent Bordeaux mixture 1:05 every three weeks with very satisfactory results and at a very considerable saving in cost of application; one can spray with 2 per cent Bordeaux mixture 1:05 every two weeks at less total expense and with results as regards protection identical with those obtained with 1 per cent Bordeaux mixture 1:05 applied weekly. But, and this is important, if we examine the data given in table 2 we will find that it is not only necessary to apply a certain amount of copper per acre per annum, but also that strictly equivalent protection is not obtained by simply making up any deficiency in strength of the Bordeaux mixture used by increasing the number of applications. The reader must not, however, infer from this that the unit copper in a strong Bordeaux mixture is more toxic than in a weak Bordeaux mixture; in other works, that there exists a relation between the strength at which the mixture is used and its toxicity to the late-blight fungus. There is no relation between concentration and toxicity, but there is a relation between composition and adhesiveness, and it is to this factor that the differences in protective power of the Bordeaux mixtures used are to be ascribed.

Now, if we turn to consideration of the proprietary fungicides employed, it will immediately be noticed that Bordeaux mixture paste, which contains when ready for use the copper equivalent of 1 per cent Bordeaux mixture, gives substantially the same protection; while Pyrox, which contains when ready for use only one-twenty-sixth of the copper present in a 1 per cent Bordeaux mixture, affords a protection that is far from satisfactory. Pyrox, it has been shown,[1] is not as adhesive as 1 per cent

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[1] Butler, O., and Smith, T. O. Relative adhesiveness of the copper fungicides, Phytopathology 40: 442, 1919.

Bordeaux mixture, so that its inability to give equivalent protection may be due both to a deficiency of copper and adhesive properties of a lower order. On the other hand, the behavior of Bordeaux mixture paste shows sufficiently that it is possible to manufacture a proprietary mixture possessing the properties of home-made Bordeaux mixture, but, in the present case at least, this was not accomplished by any economy in the use of copper. This fact should be especially borne in mind.

## SUMMARY

1. The amount of copper that must be applied per acre per annum in order to prevent losses due to the attacks of *Phytophthora infestans* lies between 24 and 26 pounds.
2. One per cent Bordeaux mixture 1: 05 is superior to 1 per cent Bordeaux mixture 1: 1 for the control of late blight.
3. Two per cent Bordeaux mixture 1: 05 applied fortnightly is superior to both 1 per cent Bordeaux mixture 1.05 and 1: 1 applied every week.
4. Of the two proprietary copper fungicides experimented with, the one giving a mixture rich in copper was found very superior to the one giving a mixture weak in copper.

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# PHYTOPATHOLOGICAL NOTES

*Puccinia graminis on native Berberis canadensis.*[1]—*Berberis canadensis,* which is native in part of the Allegheny region of the eastern United States, has been known for some time to be susceptible to *Puccinia graminis.*[2] However, naturally infected bushes have not been reported heretofore, with one possible exception. An infected specimen of what was labeled *B. canadensis* was found by Dr. Arthur in the Schweinitz collection.[3] But, after carefully examining the collection, Dr. Arthur inclined to the opinion that the specimen was *B. vulgaris.*

During the summer of 1919 the Office of Cereal Investigations of the Bureau of Plant Industry, United States Department of Agriculture, coöperating with the states in which the work was done, made a barberry and rust survey of most of the states contiguous to the barberry eradication area. The object of the work was to ascertain the rôle of barberries in developing and spreading rust in those states in which the situation was little known. Particular attention was directed to a study of the possible relation of escaped and native barberries to rust development on cereals.

*Berberis canadensis* was found naturally infected in southern West Virginia and in Virginia. The junior writer found infected bushes in Monroe County, West Virginia, on May 7. The first infected bushes found in Virginia were observed by Professor Fromme and Professor Massey, May 18, near Blacksburg, Montgomery County. Later, infected bushes also were found in Pulaski, Wythe, and Smyth counties.

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[1] The information contained in this note was accumulated by various people. The work was done cooperatively between the states involved and the Office of Cereal Investigations, Bureau of Plant Industry, United States Department of Agriculture. The writers are under particular obligation to the following men for assistance and observational work: Dr. F. D. Fromme and Prof. A. B. Massey, Virginia Polytechnic Institute, Dr. N. J. Giddings, West Virginia Agricultural Experiment Station. W. N. Christopher, formerly field assistant, Office of Cereal Investigations, did much of the survey work in West Virginia, and Dr. C. A. Ludwig, formerly assistant pathologist, Office of Cereal Investigations, found one of the rust locations in Virginia.

[2] Levine, M. N. The epidemiology of cereal rusts in general and of the black stem rust in particular. Mimeographed paper, 1919.

[3] Arthur, J. C., and Bisby, G. R. An annotated translation of the part of Schweinitz's two papers giving the rusts of North America. Proc. Amer. Phil. Soc., 57: 225–226, 1918.

It was quite evident that infection had occurred only a short time before May 7 in West Virginia, since most of the aecia were still immature. Nevertheless a few leaves were rusted in nearly every clump of bushes examined. Within a few weeks the infection became more general and more severe. During late May and early June thousands of heavily infected bushes were found in Monroe, Greenbrier, Summers, and Mercer counties, West Virginia. In fact, the bushes were so numerous that farmers were attempting to destroy them as weeds. An additional reason for the eradication of the bushes soon became clearly apparent. They were very numerous along fences, roadsides, and even along the edges of fields. It was evident, therefore, that the rust was likely to spread to the adjacent wheat fields.

By June 1 the rust had already spread from the bushes to grains and grasses in West Virginia. Many local rust epidemics began to develop by the middle of June, and, before harvest time, they reached considerable proportions. Very clearly many serious rust attacks were due to the proximity of rusted *B. canadensis* to wheat fields. In fact, there can be no question but that the presence of these bushes was detrimental to wheat growing in those regions in which observations were made in 1919. Several biologic forms of *P. graminis* undoubtedly were present, since, in addition to the common cereals, several wild grasses became infected. Apparently the rust spread only slightly from the barberries to wheat in Virginia, and no appreciable damage resulted. This may have been due partly to the early maturity of the wheat crop.

The association of the *B. canadensis* bushes with limestone formations was quite striking. Most of the bushes were small. This was very probably due to the fact that abundant seed apparently had been produced and the resulting seedlings were so badly crowded as to inhibit their normal development. The inaccessibility of the regions in which the observations were made probably accounts for the fact that the rust had not been reported on *B. canadensis* previously.

E. C. STAKMAN AND L. J. KRAKOVER.

*Sweet potato stem-rot and tomato wilt.*—The belief prevails in some sections of Maryland that tomatoes following sweet potatoes in the rotation become infected with wilt, the supposition being that the stem-rot of sweet potatoes and the wilt of tomatoes is caused by the same organism. That this is hardly likely would seem evident from the characteristic differences between the two organisms.

In view of the suspicions of the farmers, cross inoculations were made with *Fusarium hyperoxysporum* and *Fusarium lycopersici* on tomatoes and sweet potatoes respectively. *Fusarium hyperoxysporum* was isolated from sweet potato plants collected in the part of Maryland where the two diseases occur and this organism was used in the inoculations. A culture of *F. lycopersici* was obtained from Mr. F. J. Pritchard who had previously proved its pathogenicity.

In 1918, cross inoculations were made with these two organisms by inserting spores and hyphae into the stems of the plants near the soil line. Check plants were made by inoculating sweet potatoes and tomatoes with *F. hyperoxysporum* and *F. lycopersici* respectively. These experiments were repeated in 1919. None of the sweet potatoes inoculated with *F. lycopersici* or tomatoes inoculated with *F. hyperoxysporum* became infected, while a considerable number of the check plants finally developed the disease. The results of these experiments show that the organisms were pathogenic to the host from which they were isolated but that the sweet potato fungus was not pathogenic to tomatoes, and vice versa. These experiments were carried out at the Arlington Experiment Farm near Washington, D. C.

L. L. HARTER AND J. L. WEIMER.

*Head smut in sorghum and maize.*—During the month of September, 1919, the writer observed in a volunteer crop of orange sorghum near Davis, California, an attack of head smut (*Sorosporium reilianum* (Kühn) McAlp.) averaging 15 per cent by plant count. The plants in all instances produced no seed in any head. The tillers were normal or almost normal in number but much reduced in diameter. Smutted plants matured earlier than smut-free plants, producing characteristically small, ravelled inflorescences. As the sorghum was cut for silage, the losses for infected plants included all the grain and fully one-half of the fodder.

The significant feature of the head-smut attack lay in the heavy infection in the volunteer field and the almost entire absence of attack in the adjacent planted field where barley had been the preceding crop. The explanation for this is given in the well known fact that this smut entirely or almost entirely makes its attack from the soil, where its spores lie viable from many months dispite seasonal disturbances. Soil infestation from bunt is common in the same region. Maize in adjacent fields was badly attacked by maize smut (*Ustilago zeae*) but no head smut was found. There is evidence that head smut has existed at Davis for a number of years.

In the peaty delta soils near Stockton the writer, at a later date, encountered head smut in a field of King Philips hybrid maize. The attack affected the maize plants, ears, and tassels much the same as in the attack in the heads of the saccharine sorghums. The grouping of the attacked plants, however, was quite distinct. The area observed in the maize field was circular and most of the plants in the area were smutted In the volunteer sorghum field at Davis, on the contrary, the affected plants were scattered irregularly throughout the field. As maize production in the delta region is comparatively new, it may be that this fungus is of recent introduction and finds favorable conditions for propagation in these damp, peaty soils, which harbor so many fungous diseases. The infested area will be kept under observation.

W. W. MACKIE.

*Personals.* Dr. Luther P. Byars has resigned his position as pathologist in charge of nematode disease investigations in the Bureau of Plant Industry, to engage in business at Marion, South Carolina.

Mr. Wm. N. Ankeney, formerly scientific assistant in plant pathology in the Office of Cotton, Truck, and Forage Crop Investigations, Bureau of Plant Industry, has resigned this position to engage in farming at Xenia, Ohio.

Mr. Paul A. Murphy, Field Laboratory of Plant Pathology, Charlottetown, P. E. I., has resigned his position as officer in charge of potato disease investigation under the Dominion Department of Agriculture, and will take up work on April first as assistant with Dr. Pethybridge in the Division of Seeds and Plant Diseases in charge of plant pathological work in Ireland. His new address will be Royal College of Science, Dublin.

Mr. Julius Matz, who for the past year held the position of assistant pathologist at the Insular Experiment Station at Rio Piedras, Port Rico, has been appointed chief of the Division of Botany and Plant Pathology at the same station.

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Spindle-sprout; mosaic; curly-dwarf.

# *ABSTRACTS OF PAPERS READ DURING THE FIRST ANNUAL MEETING OF THE CANADIAN BRANCH, AMERICAN PHYTOPATHOLOGICAL SOCIETY, ONTARIO AGRICULTURAL COLLEGE, GUELPH, ONTARIO, DECEMBER 11, 12, 1919. R. E. STONE, SECRETARY-TREASURER*

*Health and disease in plants.* F. L. Drayton

A plea for greater study of the host in plant pathological relationships. The importance of knowing the substances in the host most easily utilized by the parasite, and a better understanding of the relationship of host to parasite. A short description of the factors of environment and the important bearing on disease control of the relationship of a plant to its environment. Types of relative and complete immunity, the importance of knowing what constitutes disease resistance, and the necessity of having a method of recognizing it, other than by infection experiments. Coöperation and coördination essential factors in plant pathology.

*Decay in the timber of pulp and paper mill roofs.* (Illustrated by lantern slides.) R. J. Blair

The wood roofs of the Canadian pulp and paper mills are usually renewed every eight or ten years on account of decay. The roofs consist of a single layer of wood 1 to 6 inches in thickness or of two planks separated by an air space. The common fault is that moist air enters the spaces between the planks, reaches the upper part of the roof, cools off in cold weather, condensing the moisture, which maintains the wood in a moist condition so that it decays.

Control. Build the roof of red cedar, ventilate the mill to carry moisture away, heat the mill with dry air, and place a heat-insulating layer on the upper side of the roof-planks.

*Butt rots of the balsam fir in Quebec Province.* W. H. Rankin

The so-called butt-rot disease of balsam fir is a complex of diseases caused by many different fungi. The most prevalent and destructive butt-rot is provisionally ascribed to *Fomes pinicola*. Butt-rot caused by *Polyporus schweinitzii* is also common. The *Fomes pinicola* (?) butt-rot occurs mixed with the butt-rot caused by *Polyporus schweinitzii*, and the earlier stages of these two decays have not been clearly separated so that they can be identified with certainty. Four or five other types of butt-rot of minor importance were studied but cannot be identified at present. In the case of the two common butt-rots, infection takes place largely through the tap root, although lateral roots are sometimes infected through wounds. There is a definite relation between the rate of growth of the balsam fir and the destructiveness of the two common butt-rots. Under conditions leading to fair or rapid growth, less than 10 per cent of the stands were found butt-rotted, and then only a negligible amount of damage was caused. Under conditions where growth is very slow, especially during the first thirty or forty years, the trees are uniformly found to be butt-rotted, irrespective of diameter. Butt-rot was found no more prevalent in regions where a heavy mortality has been experienced during the last ten years, following the bud-worm, than it is in other regions. The same marked relation between rate of growth and prevalence of butt-rot held in both cases. Butt-rot was found to be in no way responsible for the heavy mortality following the bud-worm infestation. The heavy mortality following the bud-worm infestation seemed to be largely due to the physiological effects from which certain trees could not recover, although they were able to keep alive a few years. The abnormal mortality was found to have about ceased in regions where the bud-worm has been absent for four years or more.

*Needle blight of the white pine.* J. H. FAULL

Needle blight of the white pine was very prevalent on a large scale in some of the white pine forests of Ontario in 1917, 1918, and 1919. Experiments and observations show that the disease is physiological and not communicable, and that it is related to root injury. Young trees under favorable conditions recover; older trees may recover, though more slowly, and a variable percentage, up to 5 per cent, of old trees die.

*Witches broom of the Canada balsam and the alternate hosts of the causal organism.* R. E. STONE

Attention was called to the wide distribution of the witches broom of the Canada balsam and its close relation to a similar disease of European firs. An account of culture work was given, showing that the rust (*Peridermium elatinum*) had for its alternate hosts *Cerastium vulgatum* L. and *Stellaria media* (L.) Cyrill, producing *Melampsorella caryaphyllacearum* (D. C.) Schroeter.

Attention was also called to the witches broom of the spruces and the work that had been done in this genus, making it seem probable that the causal organism belonged to the same species.

An appeal was made for viable material from the spruces in order to test out more thoroughly the relation between the witches brooms on these plants related to the balsam.

The paper was illustrated with charts, photographs, and material.

*Some comparative observations upon the shape of the basidia and the mode of spore discharge in the Uredineae and the Hymenomycetes.* A. H. REGINALD BULLER

Throughout the Uredineae and Hymenomycetes the spores produced upon the basidia are violently discharged.

Dietel's observations (1912) on the violent discharge of the basidiospores of the Uredineae have been confirmed and extended to *Puccinia graminis*. Especial attention has also been paid to spore discharge in *Puccinia malvacearum* and *Endophyllum euphorbiae-sylvaticae*. The basidia of the grain rusts represented in Eriksson's Fungoid Diseases of Agricultural Plants (1912) were all abnormal in form.

In both Uredineae and Hymenomycetes, just before the discharge of a basidiospore, a drop of fluid is excreted where the spore is attached to the sterigma. The drop varies in size, according to the species, from one-third to one whole diameter of the spore. On discharge the drop is carred with the spore. Sometimes the drops may become abnormally large, and then discharge may not take place.

In many Uredineae the basidia are curved and the sterigmata are then placed on the convex outer side of the basidium. This causes the basidiospores to be directed toward an open space, with the result that, when discharge takes place, the spores are shot away so that they escape into free air. The curvature of the basidia of the Uredineae is therefore fraught with a beautiful physiological significance.

This communication was illustrated with models of straight and of curved basidia and with lantern slides.

*The pseudorhiza of certain saprophytic and parasitic Agaricineae.* A. H. REGINALD BULLER

The pseudorhiza is an organ which is developed from the base of the stipe in a number of saprophytic or parasitic Agaricineae, e.g., in *Collybia radicata* and *Coprinus macrorhizus.* It is known to field mycologists as a *rooting base.* It is developed on the fruit-bodies when these originate at the surface of buried roots, etc., and it is negatively geotropic. It serves to push up the rudiments of the pileus and the aerial stipe to the surface of the ground where they can expand without mechanical hindrance. Light entirely inhibits its development, so that the length of a pseudorhiza is always directly proportional to the thickness of the dark substratum which must be penetrated. Illustrated with lantern slides.

*A smut of western rye grass.* W. P. FRASER

This paper describes the morphology and life history of a smut of western rye grass (*Agropyron tenerum* Vasey) which was found to be widely distributed in the prairie provinces of Western Canada. The ordinary formalin treatment of the seed gave perfect control.

*Some observations made in inspecting for leaf roll and mosaic.* J. E. HOWITT

During the past two seasons (1918–1919) an effort has been made to improve the potato crop in southern Ontario. This involved disease surveys of the potato growing sections of southern and northern Ontario; a system of inspection and certification for disease in seed potatoes; and the testing for disease and yields of potato seed grown in northern Ontario, southern Ontario, and New Brunswick.

The seed from these sources was grown side by side on eighty different plots in forty counties of the Province during the past two years, and records kept of the percentage of serious disease and yield per acre. The results obtained and the observations made during the carrying on of this work may be summarized as follows:

Seed potatoes grown in northern Ontario are freer from leaf roll and mosaic and give larger yields per acre than those grown in southern Ontario and New Brunswick. Leaf roll is especially prevalent and severe in the southern and southwestern portions of southern Ontario. In most sections of southern Ontario, leaf roll appears to spread and cause more loss each year that the same seed is used. In northern Ontario, leaf roll does not seem to spread, nor to increase appreciably in amount from year to year in the same stock. Under climatic conditions such as we have in southern Ontario, mosaic appears to be suppressed some years; that is, when seed from mosaic stock is planted in some seasons, no mosaic is observed in the resulting crop. In northern Ontario there appears to be no such suppression of mosaic; it apparently appears in infected stock year after year. Mosaic evidently spreads in the one year from diseased to healthy plants and one of the chief agents in its transmission appears to be the leaf hopper (*Empoasca mali* Le Baron).

*New or little-known diseases of potatoes which cause "running-out" of seed.* (Illustrated.) PAUL A. MURPHY

Two diseases are described the effects of which are comparable to the worst types of leaf roll or curly dwarf.

Plants affected by the first disease, called provisionally "crinkle," are dwarfed, light green, bushy, or somewhat upright. The leaves are strongly corrugated (but not spotted), curve downwards at tips, and brittle. Affected plants are easily distinguishable from leaf roll, curly dwarf, and mosaic.

The second disease, called "leaf drop," causes extreme dwarfing, gradual death of the leaves from below upwards, and premature death. It may be identical with Appel's bacterial ring disease, and with a trouble described by F. C. Stewart. There are indications that both diseases are of European origin. Their distribution on this continent is unknown.

*Plant diseases and fungi comparatively new or rare in Ontario.* R. E. STONE AND J. E. HOWITT

New stem rot of tomatoes due to *Botrytis* sp. This disease was discovered in February, 1919. It differs from timber rot by the more extensive lesions and their wider distribution. It does not form sclerotia readily. Infection takes place only under excessively humid conditions and so far the disease has been discovered only in poorly ventilated greenhouses.

Blight of tomato seedlings due to *Phytophthora infestans* (Mont ) de Bary. This disease was present in poorly ventilated seed beds 1917–1918.

Wilt of egg plant due to *Nectria ipomoeae* Hals. was widespread this last summer and was observed from Sarnia to Ottawa. It was worst on land where egg plant had been previously grown.

Potato wilt due to *Verticillium alboatrum* McA. is quite common in southern central Ontario, and is easily mistaken for Fusarium wilt. In the field it may be distinguished from Fusarium wilt by the lesions which occur high up on the stem, and the tubers, although showing internal browning, seldom rot. The disease is common on Scotch varieties, especially Davies Warrior.

Bean mosaic was not seen until two years ago, but in 1918 was widespread in the province, occurring as scattered plants.

Cucumber mosaic is met with occasionally but has not yet become prominent. It should be watched for.

Leaf spot and shot hole of cherries and plums are very common, and last spring the ascogenous stages were collected at Guelph. *Coccomyces hiemalis* Higgins occurred on cherry and *Coccomyces prunophorae* Higgins on plums.

*Exoascus unilateralis* PK. was collected last year on *Prunus virginiana* L.

Strawberry leaf blotch or scorch, due to *Marssonia potentillae* (Desm.) Fisch, seems to be widespread but often confused with the leaf spot. *Marssonia potentillae* (Desm ) Fisch was followed by *Mollisia earliana* (Ell. and Ev.) Soci.

Snapdragon rust (*Puccinia anthirrini* Diet. & Holi.) continues to be troublesome. The disease was introduced on cuttings four years ago. It does not appear to be carried on the seed nor to winter over on old plants outdoors, but is carried over in the greenhouse.

Rust of statice (*Uromyces limonii* (D.C.) Lev.) was sent in the last year.

For the past two years barley has been badly attacked by leaf spot and streak (*Helminthosporium sativum* (P.) K. & B (P. 111 & 13) and Hl. *teres*. Sacc ).

Barley has also been attacked by a stem disease, the fungus appearing like a Fusarium but the spores have not been seen. In some varieties the loss has been as high as 20 per cent.

Oats have also been attacked by *Helminthosporium avenae-sativae* (Briosi & Cav.) Lind.

Blue-grass in some counties presents a stunted and rusted appearance. A fungus *Leptosphaeria culmorum* Awd. is present.

Witches broom of black spruce due to the dwarf mistletoe (*Arceuthobium pusillum* Pk.) is present in our local swamps.

Killing of shade trees with kerosene at Hamilton in 1919. A number of shade trees were found to have been girdled by the placing of a band soaked in kerosene about the trees.

*Napicladium Tremulae. A new disease of the poplar.* A. W. MCCALLUM

This disease, which is well known in Europe, is recorded for the first time on this continent. It is characterized by the death of young shoots, which are bent double, become darkened in color and dried out, and the discoloration, drying out, and shrivelling of the leaves. The causal organism is one of the Pleosporaceae, *Didymosphaeria populina* Vuill. Only the conidial stage *Napicladium Tremulae* (Frank) Sacc. has been observed as yet. This disease is common in Ontario and Quebec and probable elsewhere throughout the range of the poplar. It has been collected on *Populus tremuloides* and *P. grandidentata.*

*Some data on peach yellows and little peach in Ontario.* L. CAESAR

The first appearance of yellows in Ontario was about the year 1874 and of little peach about 1895. Great outbreaks of yellows seem to have centered around the years 1881 and 1891. Another combined outbreak of yellows and little peach, but chiefly the latter, reached its height about 1911. In that year a new system of training inspectors was introduced, and as a result of the careful work done, the number of diseased trees was decreased from 60,000 in 1911 to fewer than 800 in 1918. The total loss from this outbreak was over $1,000,000. Experiments were conducted to determine possible methods of distribution of the diseases, and the following results were obtained:

1. The use of pits from diseased trees apparently is not a factor in distribution; for, though approximately 8 per cent of such pits will grow, if taken from trees still vigorous but clearly diseased, yet out of more than 100 seedlings thus obtained, none developed symptoms of disease. All these seedlings were kept for three years and 13 of them for six years.
2. Buds from affected trees inserted into healthy seedlings and trees produced disease in the majority of cases but not in all, the exceptions being possibly due to some of the buds being taken from very vigorous water-sprouts. Very few of the budded trees and seedlings—not over 1 per cent—showed symptoms of disease at the end of twelve months; most of them required from twenty-one to twenty-four months, and a few even thirty-four to thirty-six months.
3. Inoculating healthy trees with sap, filtered and non-filtered, from crushed diseased fruit, leaves, and twigs did not produce disease. (This experiment should be tried again by a different method.)
4. Rubbing the bark of diseased branches against the bark of healthy trees until both barks were badly ruptured did not produce the disease, even after four years' interval.
5. The presence for several years of diseased trees in an orchard does not, under some conditions at least, cause the neighboring trees to become diseased; for 5 trees, which, as the result of budding inoculation, showed disease clearly in 1914, were left in an orchard until they were dying; yet up to the present only one other tree in the orchard, and this one far removed from the affected trees, has become diseased. Orchard inspection however, and the experience of the past, tend to show that the presence of diseased trees is at times fraught with great danger and that their prompt removal is a safeguard against much loss when a general outbreak of either disease occurs.

*Inheritance of disease resistance in the common bean.* G. P. McROSTIE

The inheritance, as indicated by crosses between resistant and susceptible bean varieties, of resistance to three common bean diseases, anthracnose (*Colletotrichum lindemuthianum* (Sacc. and Magnus) Briosi and Cavara), dry root rot (*Fusarium phaseoli* (Martii) Burk), and mosaic, was reported on. Resistance to anthracnose was found to be dominant over susceptibility, while with the other two diseases, susceptibility was partially dominant over resistance. The segregation of resistant and susceptible plants in the second and subsequent generations followed definite Mendelian ratios. A number of desirable commercial types have been isolated which show resistance to the three diseases mentioned, and an attempt is being made to combine resistance to all three diseases in individual plants of various types.

*Abstract of talks given by* E. C. STAKMAN *at the meeting of the Canadian Phytopathological Society, at Guelph, Ontario, December 11 and 12, 1919*

The general subject of the discussion was "Recent Progress in the Investigation of Cereal Rusts."

The losses from cereal rusts have been enormous during the past several years. Quite evidently no adequate control measures were known. Extensive observations and investigations have been under way during the last four years in an attempt to ascertain more accurately the exact facts in the life history of the cereal rust fungi.

The results so far have shown rather clearly that cereal rusts overwinter in the uredinial stage in the southern part of the United States; but in the northern part of the country, while the leaf rusts overwinter readily in the uredinial stage, stem rust does so only rarely.

Extensive observations indicate plainly that a tremendous amount of stem rust infection in the spring comes from barberry rust. Control measures, therefore, have included an extensive barberry eradication campaign and careful cultural methods, including the destruction of weed grasses in so far as possible, careful fertilization, and the selection and development of rust-resistant varieties of grains. The task of developing resistant varieties is difficult. This is due to the fact that varieties resistant to one rust are not necessarily resistant to another. Furthermore there are so many biologic forms of stem rust on varieties of wheat that breeding becomes a regional problem.

Little progress has been made in the attempt to determine the real basis for resistance. Facts of such fundamental importance could best be obtained in a Phytopathological Institute such as the American Phytopathological Society is now attempting to establish.

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PHYTOPATHOLOGY

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# PERONOSPORA IN TURNIP ROOTS

MAX W. GARDNER[1]

WITH PLATE XII

Among turnips in storage and in the local market in Lafayette, Indiana, an occasional root was found in February, 1918, showing a dark discolored internal region extending down into the root from the crown, which appeared in cross section as shown in plate XII, figures A and B. The affected tissue varied in color from a light brown (fig. B) to a distinct black (fig. A) and was firm and quite turgid. One of these affected roots was cut and placed in a damp chamber in the refrigerator. Upon the cut surfaces of the discolored tissue there occurred a luxuriant development of conidiophores and conidia of *Peronospora parasitica*.

Subsequent tests showed that under such conditions the fungus would sporulate abundantly on a freshly cut surface of the affected tissue within twenty-four hours. Upon prolonged incubation in the refrigerator, the sporophores were produced in abundance on what was previously normal tissue surrounding the discolored region. This indicated that the fungus was enlarging its area of invasion. In one case, all of the cut surfaces of a root bore the aerial fungous growth after three weeks incubation, indicating that the infestation of the tissues had proceeded to completion. Such fresh invasion caused only a very light brown mottling and streaking of the tissues, not nearly so marked a discoloration as was found in the freshly collected specimens. In freshly cut specimens there were indications that the cortical tissues were not as readily invaded as the central cylinder (pl. XII, fig. B). The affected tissue was found to be predisposed to rotting by Rhizoctonia and the soft rot bacteria.

Microscopic examination of razor sections of the affected tissue revealed the presence of extremely large, club-shaped haustoria in the host cells. Invaded cells are discolored. From the distribution of these discolored cells, as well as the gross appearance of the tissue, it is evident that the infection extends in strands of various shapes and sizes interspersed with normal tissue.

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[1] Contribution from the Botanical Department of Purdue University Agricultural Experiment Station, Lafayette, Indiana.

In order to ascertain the relation of the parasite to the host tissue, blocks of diseased tissue were fixed and embedded in paraffin. Sections 15 μ thick stained with safranin, gentian violet, and carbol fuchsin proved very satisfactory. From the rather scanty intercellular mycelium, large, club-shaped, usually dichotomously lobed haustoria are produced within the host cells (pl. XII, figs. C and D). In many instances the haustoria are much branched, and almost or completely fill the lumen of the host cell. No enlargement nor multiplication of host cells is caused. No oospores have been found.

The spores produced on the cut surfaces of the roots in the refrigerator germinated in tap water very readily at refrigerator temperatures of 8° to 12°C., less vigorously at 19°C., and, at 23° to 25°C., only a very low percentage germinated, of which the germ tubes at twenty-four hours were only about one-third as long as those produced at the lower temperatures. At 29°C. no germination occurred.

With a suspension of these spores, a series of atomizer inoculations was made on turnip, rutabaga, and radish seedlings. These plants were held three days in a root cellar at 10°C. and then removed to the greenhouse. In another series the inoculum was rubbed on the cotyledons of turnip seedlings which were incubated in the refrigerator four days, and then removed to the greenhouse. At the end of two weeks, five out of twelve turnip seedlings of the last series and two out of eleven turnip seedlings of the first series were infected and showed abundant sporulation of Peronospora on the cotyledons. No infection occurred on the rutabagas and radishes nor on uninoculated turnips and radishes used as controls. An unsuccessful attempt was made to transplant the fungus to a sound turnip root by inserting a block of diseased tissue into a freshly made cavity of similar size and shape which was then sealed with paraffin. Microscopic examination showed that the fungus had not invaded the sound tissue in contact with the inoculum.

Melhus[2] found *Peronospora parasitica* living over winter by means of a perennial mycelium in *Lepidium virginicum*. The occurrence of viable mycelium of this fungus in turnip roots late in the winter indicates that it may also live over winter in turnips which remain in the field.

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[2] Melhus, I. E. Perennial mycelium in species of Peronosporaceae related to Phytophthora infestans. Jour. Agr. Res., 5: 59–69. Pl. III, 1 fig. 1915.

PLATE

## PLATE XII

Fig A Cross section of a turnip showing blackened tissue infected with *Peronospora parasitica*

Fig B Similar to Fig A, showing another type of lesion brown in color

Fig C Photomicrograph of a section of turnip root tissue infected with *Peronospora parasitica*, showing the intercellular mycelium and the club shaped haustoria within the host cells (Magnified about 250 times )

Fig D Similar to Fig C (Magnified about 210 times )

GARDENER PERONOSPORA IN TURNIPS

# EXPERIMENTS UPON FORMALDEHYDE-DRIP CONTROL OF ONION SMUT

J. C. WALKER

Since the increasing importance of onion smut (*Urocystis cepulae* Frost) in southeastern Wisconsin has necessitated the use of a disinfectant with the seed in certain areas to insure a crop, the experiments reported herewith were undertaken to determine the most effective means of control. In 1913, when the first experiments were carried out, both the sulphur and lime method described by Sirrine and Stewart[1] and the formaldehyde-drip treatment devised by Selby[2] were tried. That season's results seemed sufficient to show the inferiority of the former method, both as to efficiency in controlling the disease and ease of proper application. Further efforts were directed during the following seasons toward the formaldehyde-drip method. The chief practical objection to the latter has been the additional load on the hand seeder made necessary when the tank containing the disinfectant is attached. Accordingly, one of the chief objects of the work was to determine the maximum concentration and minimum quantity of the solution possible for most effective control of the disease.

## PLAN OF THE EXPERIMENTS

All of the experiments from 1913 to 1916, inclusive, were performed on the same plot of ground. This consisted of an area 475 by 50 feet, selected from a badly diseased field in the Racine, Wisconsin, district. The ground was nearly level, the soil was a uniform, black, sandy loam, and the disease proved to be very evenly distributed. Rows were run lengthwise of the field, and the series of treatments was repeated two or more times in order to compensate as much as possible for any slight variations in the soil. The Red Globe variety of onion was used throughout. Seed was planted one-half to one inch below the surface, by means of a standard Planet Jr. onion seeder, at the rate of 3 to 4 pounds per acre, which is customary in this section for the growing of large bulbs.

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[1] Sirrine, F. A., and Stewart, F. C. Experiments on the sulphur-lime treatment for onion smut. N. Y. (Geneva) Agr. Expt. Sta. Bul. 182, p. 143–172. 1900.

[2] Selby, A. D The prevention of onion smut. Ohio Agr. Expt. Sta. Bul. 131, p. 47–51. 1902.

The drip attachment has been changed from time to time during the course of the experiments, and since the type in present use has been fully described elsewhere,[3] it need not be considered further at this piont.

## DISCUSSION OF RESULTS

Since obviously many factors other than the disease influence the crop in an experiment of this sort, it is not advisable to depend upon yield alone in comparing the efficiency of various treatments. The percentage of smutted seedlings seems to be a much better criterion on which to base judgment in this case. Throughout the experiments the amount of disease was estimated from time to time. This was usually done by measuring off four 20-foot strips crosswise of the plat at equal intervals and counting healthy and smutted plants in these portions of each row. During 1914 and 1915 such counts, made approximately one, two, and three months after planting, showed that in both seasons the maximum amount of disease in all the treated plots was secured at the second count, that is in the last part of June. The decline in the percentage of diseased plants after this date was due to the death of infected seedlings. In the results summarized in table 1, counts made at this time of the season are given, except for 1913, when the first counts were not secured until August.

In 1913, three strengths of formaldehyde were used, namely, ½ liquid ounce of the concentrated solution (37 to 40 per cent) to one gallon of water (1:256), 1 ounce per gallon (1:128), and 2 ounces per gallon (1:64). It is clear from the results given in table 1 that the strongest solution gave the best control, as indicated by both the amount of smut and the final yield, while the weakest solution had practically no effect on the disease.

In 1914, the weakest solution was dropped from the schedule, while a stronger one (1:32) was added, and, in addition, the amount of liquid was varied (118 and 236 gallons per acre). In the case of both quantities the 1:32 solution caused a reduction of stand, and, moreover, the control of the disease was no more complete than in the case of the 1:64 solution. The low yield in the case of the 1:64 solution applied at 118 gallons per acre could not be satisfactorily explained, but it is evidently not due to seedling injury, since the same strength at 236 gallons gave a much higher yield. Considering the percentage of disease, the latter strength at 236 gallons is the best of the series, although the 1:128 solution at 236 gallons per acre and the 1:64 solution at 118 gallons gave nearly as good results.

In 1915, attention was confined to the 1:128 and 1:64 solutions applied at 118 and 236 gallons per acre. As indicated in the previous season, the disease was controlled much more effectively when the larger quantity was applied, and in this case the weaker solution (1:128) gave as satisfactory results as the stronger (1.64).

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[3] Walker, J. C. Onion diseases and their control. U. S. D. A. Farmers' Bul. 1060, p. 1–24. 1919.

In 1916, experiments parallel with those carried out by Dr. I. C. Jagger in New York were planned, and consequently the details of treatment

TABLE 1

*Summary of results of formaldehyde control experiments. Racine, Wisconsin, 1913–1916*

| YEAR | DATE OF SOWING | NUMBER OF PLOTS USED | NUMBER OF ROWS PER PLOT | STRENGTH OF SOLUTION | NUMBER OF GALLONS PER ACRE | AMOUNT OF SMUT | | FINAL YIELD IN BUSHELS PER ACRE |
|---|---|---|---|---|---|---|---|---|
| | | | | | | Date of observation | Per cent | |
| 1913 | April 22 | 5 | 2 | Untreated | | August 6 | 24.0 | 325.2 |
| 1913 | April 22 | 2 | 2 | 1–256 | 118± | August 6 | 27.5 | 340.8 |
| 1913 | April 22 | 2 | 2 | 1–128 | 118± | August 6 | 5.6 | 505.9 |
| 1913 | April 22 | 2 | 2 | 1–64 | 118± | August 6 | 2.5 | 588.3 |
| 1914 | April 23 | 5 | 2 | Untreated | | June 30 | 52.0 | 86.4 |
| 1914 | April 23 | 2 | 2 | 1–128 | 118± | June 30 | 13.4 | 344.4 |
| 1914 | April 23 | 2 | 2 | 1–64 | 118± | June 30 | 10.3 | 282.9 |
| 1914 | April 23 | 2 | 2 | 1–32 | 118± | June 30 | 11.5 | 215.8 |
| 1914 | April 23 | 2 | 2 | 1–128 | 236± | June 30 | 10.2 | 295.9 |
| 1914 | April 23 | 2 | 2 | 1–64 | 236± | June 30 | 6.2 | 409.2 |
| 1914 | April 23 | 2 | 2 | 1–32 | 236± | June 30 | 6.3 | 298.8 |
| 1915 | April 14 | 3 | 2 or 4 | Untreated | | June 25 | 52.5 | 400.6 |
| 1915 | April 14 | 2 | 2 or 4 | 1–128 | 118± | June 25 | 28.5 | 633.6 |
| 1915 | April 14 | 2 | 4 | 1–64 | 118± | June 25 | 15.3 | 721.6 |
| 1915 | April 14 | 2 | 4 | 1–128 | 236± | June 25 | 4.3 | 746.7 |
| 1915 | April 14 | 2 | 4 | 1–64 | 236± | June 25 | 3.5 | 654.2 |
| 1916 | April 24–27 | 10 | 1 | Untreated | | June 19 | 54.9 | 72.9 |
| 1916 | April 24–27 | 3 | 1 | 1–175 | 70–100 | June 19 | 43.1 | 117.1 |
| 1916 | April 24–27 | 4 | 1 | 1–125 | 81–100 | June 19 | 29.0 | 163.8 |
| 1916 | April 24–27 | 4 | 1 | 1–75 | 80–107 | June 19 | 13.7 | 177.5 |
| 1916 | April 24–27 | 4 | 1 | 1–175 | 128–150 | June 19 | 31.4 | 181.8 |
| 1916 | April 24–27 | 4 | 1 | 1–125 | 134–150 | June 19 | 24.2 | 207.4 |
| 1916 | April 24–27 | 4 | 1 | 1–75 | 131–156 | June 19 | 14.6 | 205.3 |
| 1916 | April 24–27 | 3 | 1 | 1–175 | 200–224 | June 19 | 17.7 | 291.5 |
| 1916 | April 24–27 | 4 | 1 | 1–125 | 197–200 | June 19 | 5.8 | 350.8 |
| 1916 | April 24–27 | 4 | 1 | 1–75 | 193–198 | June 19 | 7.1 | 247.0 |

were changed slightly. The desirability of applying larger amounts was again brought out, and when approximately 200 gallons per acre were used, the 1: 125 strength gave the best control. Experiments in which the quantity was increased to more than 236 gallons were not performed, since this represents the approximate limit which can be practically handled on a common hand seeder, and the control is complete enough for general purposes when this amount is used.

It may be concluded from these data that for the section concerned at least, approximately 200 gallons of solution per acre will give the most satisfactory results, and that there is no advantage in this case in increasing the concentration above 1:128 (1 liquid ounce per gallon or 1 pint to 16 gallons). Where there is serious objection to using this amount of liquid because of difficulty in application, the quantity may be reduced to 120 to 150 gallons, but in this case the concentration should be increased to 1:64 (2 liquid ounces per gallon). Raising the strength of the solution beyond this point, however, entails danger of reduction of stand due to seedling injury.

It is very probable that numerous environmental factors, such as type of soil, temperature, and moisture, influence more or less the effect of formaldehyde upon both the fungus and the host plant. Some of these perhaps have significant practical bearing and should be considered in making general recommendations for a wide area on the basis of experiments in one locality. The conditions following sowing also vary from year to year in the same locality. For instance, there was rainy weather immediately after the experimental plots were planted in 1914, while in 1915 no rain fell for eighteen days after sowing was completed. In the former year there was little or no retardation of seed germination in the treated plots, while in the latter all the treated plots were distinctly slower in starting off than were the checks. Moreover, in 1915, where the smaller quantity of solution was used, the control of the disease was much less pronounced than in similar plots the previous year.

TABLE 2

*Results of commercial application of 1:128 formaldehyde for the control of onion smut*

| YEAR | GALLONS PER ACRE | PER CENT PLANTS DISEASED | | YIELD, BUSHELS PER ACRE | |
|---|---|---|---|---|---|
| | | Treated | Untreated | Treated | Untreated |
| 1916 | 130 | 15 0 | 43 5 | 328 | 203 |
| 1917 | 200 | 2 3 | 52 2 | 544 | 200 |
| 1918 | 200 | 2 5 | 50 0 | 682 | 186 |

However, it is not believed that this or other factors are influential enough to seriously limit successful control of the disease by this method in the section concerned, and probably the same statement holds for other northern onion-growing regions. As further substantiation of this tentative conclusion, data are cited in table 2 which were taken for a period of three years from a badly diseased field in the Racine district, where a 1:128 formaldehyde solution was applied.

In sowing onion seed for sets, as they are grown in the Wisconsin, Illinois, and Indiana sections by the single row method, about twenty times as much seed is used per row as in the case of large bulb production. This fact does not necessarily alter the quantity of formaldehyde solution to be applied, since the same amount of soil surrounding the seed is to be disinfected in either case. Furthermore, results secured with the use of the above formula by onion set growers in both the Racine and the Chicago districts indicate that it is satisfactory for use with this crop. With the multiple-row system, in use in certain onion sections, a different formula will be necessary.

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# A NEW CORN SMUT IN WASHINGTON

B. F. Dana and George L. Zundel

With Four Figures in the Text

*Occurrence.* In the fall of 1919, a smut of corn (*Zea mays* L.) not previously known to occur in Washington was found by the writers in the vicinity of Pullman. The symptoms and microscopic characters of the disease showed that it was not the common or boil smut of corn but was identical with the head smut of sorghum caused by *Sphacelotheca reiliana* (Kühn.) Clinton.

Two widely separated fields were found to be affected, one field showing 40 per cent of smutted plants. "Thayer's yellow dent" corn was the variety grown in each case, the seed having been selected from locally grown stock for a number of years. One grower stated that smut occurred on his corn "every year" but it cannot be said with certainty that head smut occurred before the present season in Washington, either on corn or sorghum, the more common host for this disease. We have no reports of the disease west of Nebraska or Texas. However, a disease having the symptoms of head smut has been seen by one of the writers in Utah on sorghum, although a definite determination of the fungus was not made. The fact that the disease has occurred, as far as is known, only on locally grown stock makes the manner of introduction difficult of explanation.

*Description.* Head smut has received full treatment at the hands of Potter[1] but most attention was given to the disease as it occurs on sorghum. Butler[2] gives a detailed description of head smut on corn as it occurs in India. Below is a somewhat detailed description of head smut on corn as it occurs in Washington. Sori or smut masses were observed in ears and tassels only. In one field tassels were most commonly affected, smutted ears being rare. In the other field the reverse was true.

The majority of the tassels showed black, powdery smut masses, involving more or less of the whole inflorescence (see fig. 1). In a few cases sterility was produced, accompanied by a distinct proliferation of the flower parts and the tassel as a whole. Figure 2 shows this development. The production of leafy shoots in the place of normal parts is clearly shown.

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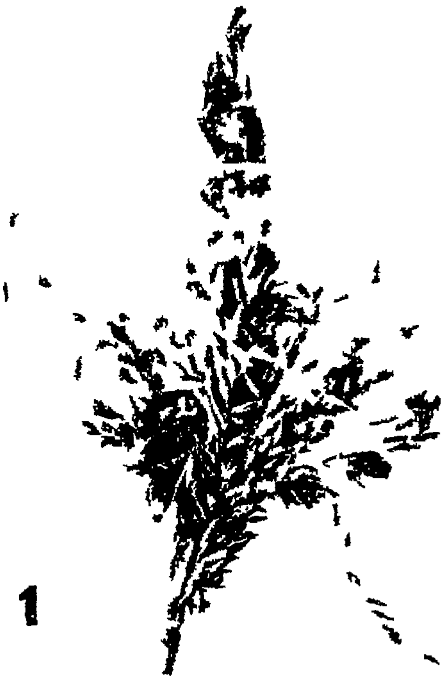
[1] Potter, A. A. Head smut of sorghum and maize. Jour. Agr. Res. 2: 339–372. 1914.

[2] Butler, E. J. Fungi and disease in plants. Pp. 199–201. 1918.



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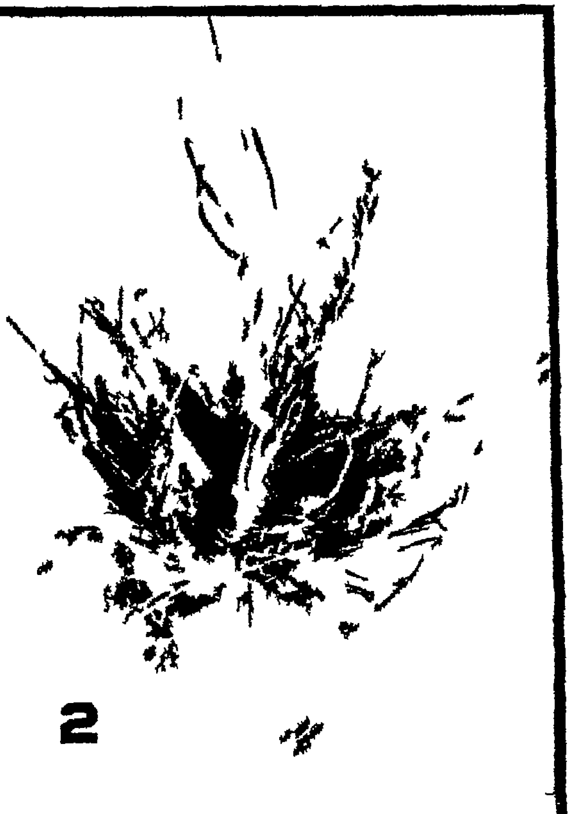

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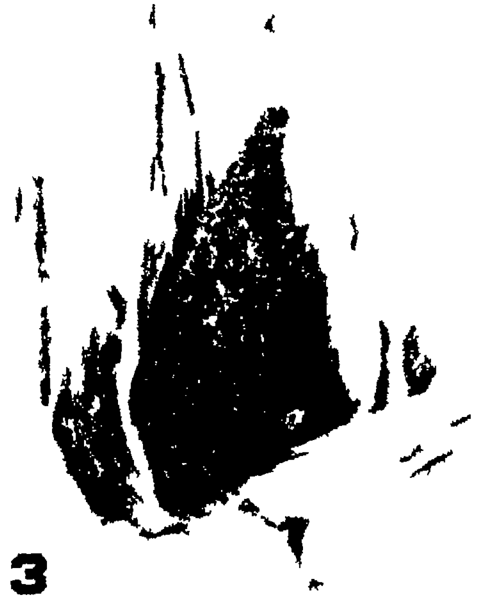

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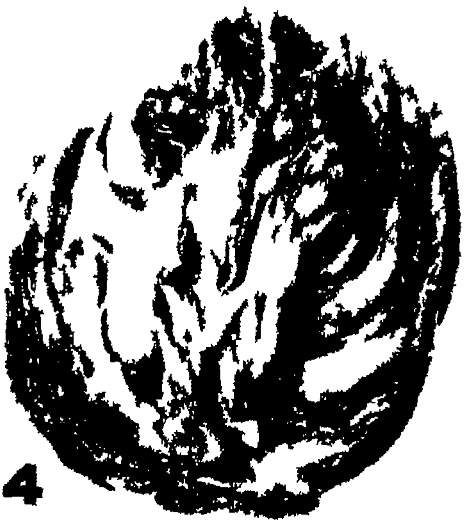

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Fig 1 Tassel Showing the Usual Effect of Head Smut
Fig 2 Tassel Showing Sterility and Proliferation
Fig 3 A Smutted Ear Showing the Characteristic Fibers
Fig 4 Sterility and Proliferation in the Ear

The only smut spores produced in this ear are to be seen in a small mass near the top of the figure

Within some of these shoots there was a mass of smut spores not apparent from the outside. Figure 3 shows the usual effect observed on the ear. The whole ear is involved and the characteristic "fibers" running through the mass are clearly shown. This character alone would be sufficient to distinguish it from the common smut caused by *Ustilago zeae* (Beckm.) Unger. A very common occurrence was the production on a smutted plant of abortive ears at the nodes below the main ear. These were usually completely smutted, some showing, however, irregular, leafy growth. In one case observed, leafy growth involved the whole ear, as shown in figure 4. The nature of this proliferation is essentially the same as was shown in figure 2 on the tassel. In a small section of one of the leafy shoots there was a small mass of smut spores. This can be seen near the top of figure 4. These figures bring out strikingly the fact that head smut may produce sori in the tassels and ears or may cause abnormal development in these parts. Affected plants were not as tall or thrifty as normal ones, and exhibited a rather uniform dwarfing effect of the fungus. Some instances were observed where a normal ear was produced on a plant showing smut in the tassel. Many plants were seen where the ear was smutted and the tassel was normal; however, in one feld where special study of symptoms was made the ear was generally affected where the tassel showed the smut masses. It is hoped that conditions will not be favorable for the spread of head smut in Washington.

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# COIX SMUT

## C. C. THOMAS

WITH ONE FIGURE IN THE TEXT

The appearance of a disease, previously unreported in this country, in the Plant Quarantine Greenhouse of the United States Department of Agriculture is of special interest to all who are concerned with the prevention of the introduction of new diseases into this country.

Coix smut (*Ustilago coicis* Bref.) has recently appeared on a plant grown from one lot of seed of *Coix lachryma jobi* sent in by Dr. W. H. Weston of the Office of Cereal Investigations from Kalasungay, Mindanao, Philippine Islands.

The pathological inspectors of the Federal Horticultural Board ordered this lot of seed grown in the quarantine greenhouse as a precautionary measure, inasmuch as it came from within the territory covered by Quarantine No. 24, which prohibits the importation of *Zea mays* and its relatives from certain Oriental countries, as a protection against the downy mildews of corn.

The seed was planted April 14. 1919, and the plants grew to a height of ten feet before flowering. The disease was first observed December 29, 1919, at which time the plants had almost finished flowering. There were four culms in the pot in which the disease appeared, only one of which was diseased. One culm did not flower, and the flowers on the other two did not set seed.

Our knowledge of the life history and distribution of this disease is very imperfect. It was first described by Brefeld,[1] his specimen having been received from Barclay of India. Butler[2] reports that it has since been collected in Bombay and Java. He further states that no information is available regarding the amount of damage caused by the disease, but that it may cause considerable loss, since it entirely destroys the grain.

There is a specimen of this disease in the Office of Pathological Collections of the United States Department of Agriculture, which was collected by Kusano in Japan in 1904.

Every grain of a diseased head is converted into a black spore mass with a slight increase in size, which in some cases may reach almost twice the size of the normal grain. The grain may be badly deformed or it may be almost normal in outward appearance as is shown by the photograph (fig. 1). The spore mass is surrounded by a membrane and is more or less

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[1] Brefeld, O. Untersuch aus d. gesammtgeb. D. Mykologie, 12, p. 110, 1895.

[2] Butler, E. G. Fungi and Disease in Plants, p. 242, 1918.

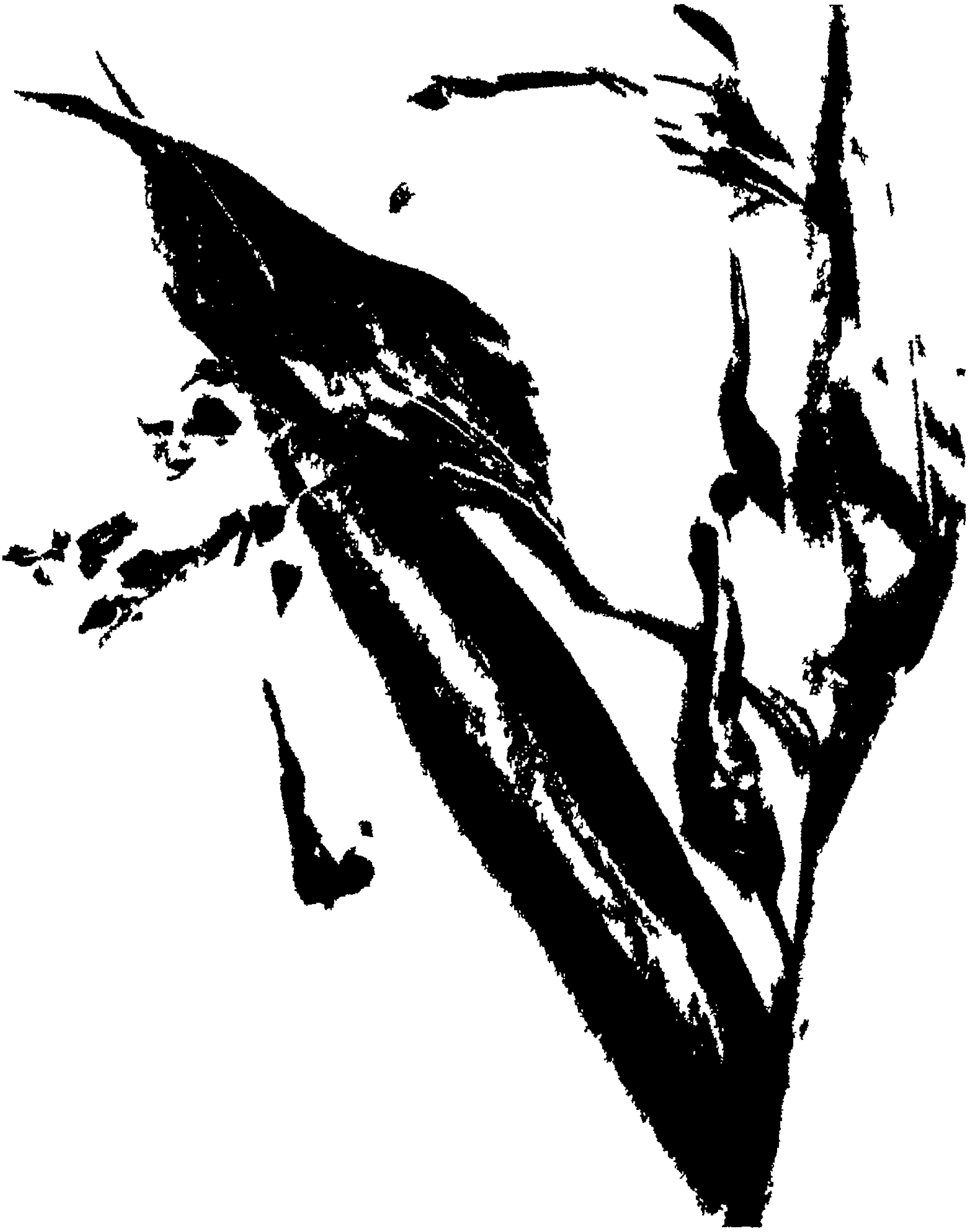

FIG. 1. SMUTTED HEAD OF COIX LACHRYMA JOBI

All grains here shown are filled with a black spore mass. Photographed by E. L. Crandall.

hidden by the glumes. The leaves and portions of the culms adjoining the diseased grains are distorted and bent. The smut also appears on the culm, as is shown very distinctly by the photograph (fig. 1).

The spores are dark brown, round or slightly oval, and have an echinulate epispore. Measurements of the spores in this material correspond very closely to those given by Brefeld (7–9$\mu$).

The spores have been germinated without difficulty on ordinary blotting paper moistened with tap water and placed in a moist chamber. A typical four-celled promycelium is produced, and terminal and lateral sporidia are produced, which in turn may bud off secondary conidia. In some cases the primary sporidia elongate into a septate filament which buds off secondary conidia, terminally or laterally, near the septa.

Brefeld describes the germination of the spores after being kept in moist earth two years and then sown in a nutrient solution, but he failed to get germination in water. Butler reports the germination of the spores in Java in water and in a nutrient solution immediately after ripening.

The life history of the causal organism is unknown and it would be difficult to say in this case whether it was introduced by spores on the seeds or by dormant mycelium within.

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# ABNORMAL GROWTH INDUCED BY CHLORAL HYDRATE SOIL TREATMENT

CARL HARTLEY

WITH ONE FIGURE IN THE TEXT

In soil-treatment experiments conducted by the writer and Mr. S. C. Bruner in western Kansas in 1914, primarily for the purpose of securing data on the damping-off disease, seedlings of western yellow pine (*Pinus ponderosa*) exhibited an unexpected reaction. A number of plots were sown in a very light, sandy soil containing little humus and practically neutral as shown by hydrogen-ion determinations by Dr. L. J. Gillespie. Two of these plots were treated with chloral hydrate in quantities of ¼ and $\frac{9}{16}$ ounces per square foot, respectively (0.076 and 0.172 kgm. per square meter). The applications were made in aqueous solution three days before sowing. A fair number of seedlings appeared above ground in all of the plots in this series by seventeen days after the seed was sown. In the five untreated plots, and in plots treated with dried blood, bone meal, ether, carbon bisulfid, sulfuric acid, formaldehyde, methyl alcohol, and ethyl alcohol, the seedlings were normal in appearance. In the chloral hydrate plots the number of seedlings appearing was only three-fifths as great as in the two nearest untreated plots (sown with equal quantities of seed and from the same lot) and many of the seedlings were strikingly abnormal in size and form. Typically abnormal seedlings from these plots are shown in figure 1, *A* and *B*; figure 1, *C*, shows a seedling of normal type for comparison. The affected seedlings were taller than normal plants, and the cotyledons and the first juvenile needles were partially fastened together, forming imperfect cylinders. The cotyledons were longer than those of normal seedlings. The loss from damping off in the chloral hydrate plots was distinctly heavier than in the untreated plots—47 per cent of the seedlings against 25 per cent in the two nearest untreated plots.

The size and form of pine seedlings during the first few weeks after germination are relatively constant. Hundreds of seedbeds of *P. ponderosa* and other conifers have been observed in different parts of the country, and following soil treatments with acids, hydroxides, salts, and organic substances. In no other case was so abnormal a development found. Occasional seedlings of both *P. ponderosa* and *P. resinosa* germinated in autoclaved soil containing compost have been found with adhesions between two or three cotyledons, which do not break up until two or three weeks after the seedlings appear. In the autoclaved soil as in the chloral hydrate plots the seedlings showing the adhesions were generally above normal size. The adherent epidermal surfaces after separation appeared normal.

It is of course impossible to say whether the striking abnormality in the chloral hydrate plots was due to a direct effect of the chloral hydrate or one of its derivatives on the seedlings, or to some indirect effect. The fact that the tests were in a coarse mineral soil suggests the possibility of a direct effect. In both the chloral hydrate plots and the autoclaved soil-compost mixture the amount of soluble organic matter was presumably above average.

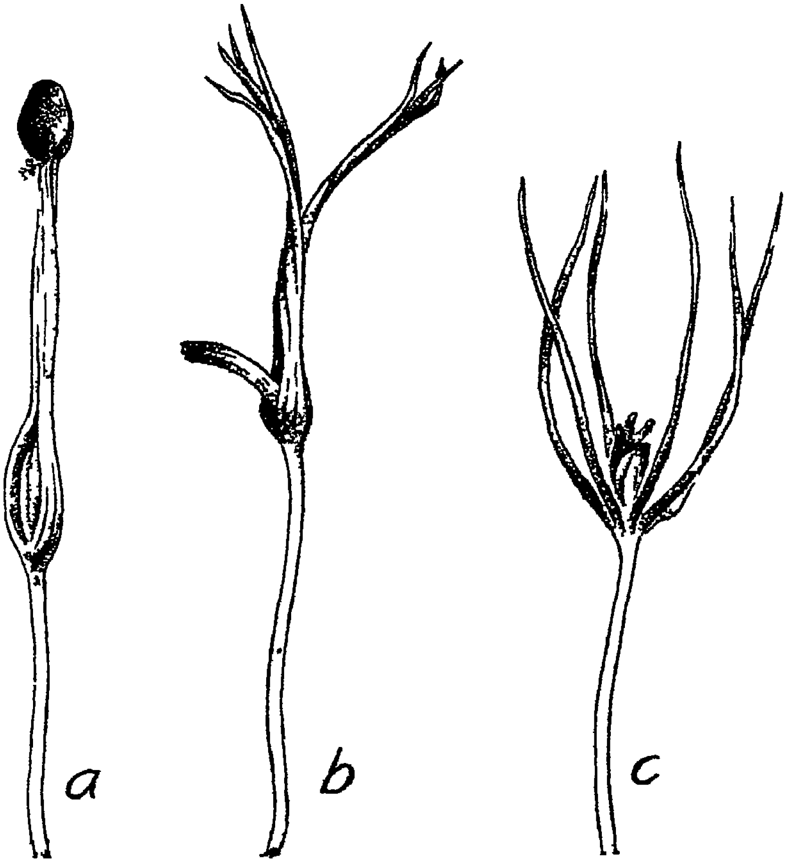

FIG. 1. SEEDLINGS OF PINUS PONDEROSA

*A* and *B*, from a seed bed which has been treated with chloral hydrate; *C*, normal type from a neighboring untreated bed. Natural size.

OFFICE OF INVESTIGATIONS IN FOREST PATHOLOGY
U. S. BUREAU OF PLANT INDUSTRY
WASHINGTON, D. C.

# A BATTERY OF CHAMBERS WITH DIFFERENT AUTOMATICALLY MAINTAINED TEMPERATURES

B. E. LIVINGSTON AND H. S. FAWCETT

## ABSTRACT

This paper calls attention to the need for a number of maintained temperature chambers that may be operated simultaneously, for studies dealing with the relations of temperature to growth, etc. The main essentials of a battery of seven such chambers, constructed in 1916–1917 at the Laboratory of Plant Physiology of the Johns Hopkins University, are described. A suitable rectangular tank is divided into ten compartments, the first being for ice and water and the second and tenth being for water, with automatically controlled electric heaters and stirrers. The seven intervening compartments contain the cylindrical culture chambers, each supplied with water-jacket and stirrer. Water does not circulate from one compartment to another, but the water of each compartment (excepting the first) is mechanically stirred. Access to the chambers is had from above and the motor-driven stirring mechanisms and heating units are connected below. Suitable insulation and an outer case for the whole series are provided.

In operation, the water of one heated compartment is kept at a lower temperature and that of the other heated compartment is kept at a higher temperature; the seven intervening culture chambers assume and maintain different temperatures between the two selected extremes. One setting had the two controlled temperatures fixed at 5° and 45°C., respectively, and the maintained temperatures of the culture chambers were 7.5°, 13.5°, 18.5°, 24.5°, 30°, 35.5°, and 40°C. After a preliminary period of less than 48 hours, during which a steady state is established, the apparatus operates indefinitely, with fluctuations in any chamber of not over 0.5°C. A table of growth rates of the fungus *Phytophthora terrestria* on corn-meal agar for the seven maintained temperatures just mentioned, is appended.

* * * * * *

The experimental study of the influence of temperature upon physiological processes requires a number of distinct chambers, each with its own controlled temperature, so that a whole series of different temperature conditions may be tested simultaneously. This need has been felt especially in connection with certain lines of study in pathology and bacteriology, as well as in many lines of physiology. Numerous makes of chambers, or incubators, with automatically controlled temperature have made their appearance on the apparatus market during the last decade, while other devices have been specially constructed in various laboratories; but considerations of expense have thus far generally prevented the simultaneous use of a considerable number of these. Aside from the matter of cost, moreover, a really satisfactory incubator usually occupies so much space that the room needed for a battery of them is frequently in default. On the whole, it may be said that, up to the present time, there are comparatively few laboratories for pathological or physiological experimentation in which as many as five chambers with reliably controlled temperature may be brought simultaneously into service.

Being impressed by the very great need for a much more thorough understanding of the physiological relations of temperature than is now available, and being convinced that productive enquiry into this fundamental field of physiological science requires, in the very first place, such a battery of temperature-controlled chambers as has just been suggested, we have attempted to satisfy our needs by constructing a series of these in the Laboratory of Plant Physiology of the Johns Hopkins University. This paper deals with some of the essentials of their construction and operation.

Chambers with temperature control may be classified primarily in two categories: (1) those in which light is allowed to act on the experimental organisms or material, and (2) those from which light is excluded. Obviously the second kind offers far less practical difficulty than the first, and the chambers to be described below were planned as dark chambers. Each of these two categories of chambers may be further subdivided into two general classes: (a) those in which the selected temperature is *maintained* for long periods of time, and (b) those in which a definite rate of temperature change is provided for by automatic controls. So far as we are aware the latter kind of apparatus has not yet been seriously attempted, though it will become essential as soon as the influence of *rate of temperature change* upon organisms begins to attract attention. Our apparatus belongs essentially to the former class. It was our aim, then, to devise and construct a battery of dark chambers, in each of which the temperature would be automatically maintained for any desired length of period. The series was to be as compact as possible, and access to each chamber was to be independent and as easy as might be. The size of chamber chosen was such that the smallest form of thermograph now on the market (the one taking record sheets 22 × 6.5 cm., made by J. Richard, Paris) might operate within the chamber and still leave plenty of room for cultures, etc.; it is highly desirable that every thermostatic chamber have its own individual thermograph. Since the consensus of experience in temperature maintenance points clearly to the employment of the water-bath type of chamber, on account of the enormous specific heat of water and the ease with which temperature differences within the same chamber may be avoided by circulating this liquid, each of our chambers is surrounded below and laterally by a considerable mass of continuously circulating water. Access to the chambers is had from above, which fact, together with other considerations, has made it impracticable to water-jacket the tops. Experience has also shown that the electric current is by far the most satisfactory source of heat for such temperature control as is here desired, and this was adopted. From our own experience, and that of many others, we determined to employ mercury-contact sensitive members in our thermostats,[1] and the type actually employed, with perfect satisfaction, is that devised by Hasselbring and manufactured by H. J. Green, Brooklyn, N. Y. These beautiful and efficient little instruments may be set to hold any temperature that may be desired in biological work, and they appear to operate indefinitely without any alteration, so that they require no attention after they have been once adjusted. This type of sensitive member far surpasses many other forms that have been described, in precision of operation and in prolonged reliability. Since our aim was to attain the desired end with as little expense as possible, and since the particular *degrees* of temperature (on any particular thermometer scale) represented in such a series of chambers are generally of no practical imimportance in experimentation—temperature being a *continuous* variable—we have employed only two thermostats for the entire battery. The temperatures of the two ends of the series are alone thermostatically controlled, and the intermediate chambers assume temperatures determined by the temperature-gradient that exists between the two ends. Finally, it is generally necessary to employ some temperatures markedly below that of the air surrounding the apparatus, so that some form of heat-absorber is requisite. We have employed the primitive method of allowing melting ice to absorb the superfluous heat at the cold end of the series.

The foundation of the apparatus is a rectangular tank of galvanized sheet-iron 4.2 m. (15 feet, 9½ inches) long, 51 cm. (20 inches) high, and 46 cm. (18 inches) wide, divided into ten conpartments by cross-partitions of the same material. The compartment at one end is the ice tank, large enough to hold ice for a run of a day or two. The second and tenth (last) compartments are small, being only 23 cm. (8 inches) in length, measured on the axis of the large tank. Each of these is supplied with two double stirrers and with a thermostat. The intervening seven compartments are each 43 cm. (17 inches) in length, being nearly square as seen from above. Each of them is provided with an internal, vertically placed, galvanized sheet-iron cylinder closed at the bottom and raised about 6 cm. (2½ inches) above the floor of its compartment. A suitable stirring mechanism is introduced through the bottom of each of these cylinders, and two stirring-rods extend upward close to the lateral wall, being bent (in inverted U-form) so as to clear the free edge and then extend downward, on the outside, as far as the bottom of the cylinder. The outer limbs of these rods are continually moving around the cylinder, circulating the water of the compartment. The water-jacket continues beneath the cylinder, so that the latter is completely surrounded by water, excepting for its open top. The whole large tank is suitably encased and is insulated with a 6.5-cm. (2½ inches) layer of plasterer's hair at sides, ends and bottom. Each compartment has a removable top, built up of several layers of thin wood and wool felt, and each of the seven culture compartments (third to ninth, inclusive) has a circular opening in its top through which is suspended a vertical lining cylinder (of multi-perforated, tinned sheet-iron), which is within the cylinder already mentioned, reaching nearly to the bottom of the latter. This lining cylinder has a bottom of perforated material but it is freely open at the top. Around and below it is air-space, and the inner limbs of the stirrer rods move between the lining and the outer cylinder, thus tending to keep the air of the culture chamber (within the lining chamber) in slow but constant motion. The lining cylinders are provided with suitable removable lids, for easy access to their interiors. Cultures, etc., are placed on wire racks fitting in the chambers.

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[1] The word *thermostat* is loosely used by many writers, to denote either (1) the sensitive member of the temperature-controlling system, (2) the whole apparatus, including the chamber walls, the controlling arrangement and the chamber space, or (3) the chamber space alone. We here employ the word in neither one of these three senses, but use it to denote the complete system that controls the heat supply. In this sense the thermostat consists essentially of three parts, the sensitive member, the heating member and the mechanism through which the former controls the heat supplied by the latter. In this sense the word becomes parallel with *klinostat* and *heliostat*, meaning the whole mechanism by which the condition considered is maintained.

The amount of water used is a very important feature in determining the constancy of the temperatures within the several chambers. The ice compartment holds about 138 liters (39 gallons), each electrically heated compartment holds about 36 liters (10 gallons), and the water-jacket space about each culture chamber holds 39.5 liters (11 gallons) of water, when all are filled to the water level determined by the overflow openings.

All stirrers are operated by a single electric motor, by means of belts and pulleys underneath the whole apparatus and thus entirely out of the way.

As has been said, thermostatic control is applied only at the two ends of the series of chambers, in the two small compartments (second and tenth), the water of each one of these two compartments being held at its own pre-determined temperature. Suitable relays are employed for the thermostats. The maintained temperature of the first directly controlled compartment is somewhat higher than that of the ice compartment. Ice is added at intervals, usually daily. The other controlled compartment (tenth) has a temperature higher than that of the one next to the ice.

Since the principle upon which this battery of chambers is constructed is that of a maintained temperature gradient between the two ends of the series, it follows that constancy in the several chamber temperatures depends upon the preliminary establishing of a dynamic heat equilibrium between the two heated compartments. It also depends, of course, on the constancy of the rate of loss or gain of heat laterally, upward and downward, through the walls of the series. These latter rates are kept nearly constant by making them as small as possible, through insulation.

When the apparatus is started, or when a change in the temperature range has been made, it requires about 24 hours (never over two days) to establish the permanent temperature gradient, and the system must be operated idle until this is accomplished. After a steady condition of temperature has been attained the apparatus operates indefinitely, with the daily addition of ice, and with a total range of temperature fluctuation in any chamber of less than half a degree centigrade.

The following statements are appended as an example, showing how the apparatus may be employed to obtain a growth-temperature curve for a fungus growing on agar plates in petri dishes. The thermostat of the compartment next to the ice compartment was set to maintain a temperature of 5°C., and the thermostat of the other heated compartment was adjusted for a temperature of 45°C. In less than 48 hours the intervening culture chambers had each assumed a practically steady temperature, as shown by the thermograph records. These temperatures were maintained, with a total range of not above 0.5°C., during a period of six days. Petri dish cultures of the fungus *Phytophthora terrestria* on corn-meal agar showed the following average daily radial growth rates (mm.) in the seven chambers respectively.

| | MAINTAINED TEMPERATURE | | | | | | |
|---|---|---|---|---|---|---|---|
| | 7.5° | 13 5° | 18 5° | 24.5° | 30° | 35 5 | 40 |
| First day | 0.02 | 1 0 | 2.5 | 4.2 | 5.5 | 4.8 | 0.0 |
| Second day | 0.15 | 2 0 | 6.3 | 10 5 | 13.8 | 4.2 | 0.6 |
| Third day | 0.21 | 2.3 | 6.4 | 11.3 | 13.3 | 2 6 | 0.0 |
| Fourth day | 0.70 | 3.2 | 8.2 | 11.4 | 13 2 | 2.5 | 0.0 |

It is seen that the minimum maintained temperature for growth was not encountered for any day of the period, but it is evident that this minimum would not have been much below 7.5°, especially for the first day. The maximum for growth clearly lay between 35.5° and 40° for all four days, apparently becoming lower with succeeding days. The optimum temperature for the growth of this organism, as here dealt with, may be considered as about 30°.

# REVIEWS

*Les Vanillieres de Tahiti et Moorea* is the subject of a 44-page report by Dr. E. P. Meinecke, published for the Chamber of Agriculture of these islands in 1916.

In the introduction it is noted that at least twenty-five fungous parasites have been reported on vanilla, many of these occurring in Tahiti and Moorea. The plantations of these islands were reported to be in a wretched condition as shown in the following: In 80 per cent of the fields, the plants were weakened by the over-production of the pods; in 90 per cent, the plants were derived from exhausted vines; 70 per cent of the plantings were more or less ruined by the chickens, the pigs and the horses; 80 per cent of the plantations suffered from too much sun or too much shade; in 90 per cent of the plants were wounds caused by the laborers; in 60 per cent, protection from the sun was neglected.

Vanilla plants grown under such conditions as outlined in this introduction would seem to be subjects proper for a horticulturist rather than for a phytopathologist, but Dr. Meinecke has treated the subject entirely in a practical way. In the first chapter he describes in detail the needs of the vanilla plant or the proper environment for it in order to avoid diseases. In the second chapter he first treats of sun scald, degeneration of the plant, and injuries, or in other words such injuries as would not happen if the plants were grown under proper conditions; then of the insects, none of which seem to be serious, but included are an aspidiotus on the leaves, stem and young fruits, a wooly plant louse (*Pucerons lanigeres*) upon the vine and upon the petioles, ants, a microlepidoptera, a curculionid, and others of less importance The parasitic diseases are then discussed; these include Cuscuta, common but not serious, the anthracnose, the most serious of all and due to *Gloeosporium vanillae* Massee, connected with other forms and given other names also but these mycological questions are not settled. The anthracnose attacks leaves, stems and fruit but does not seriously injure vanilla grown under the most favorable conditions. The author, however, does not go into detail as to what conditions are most favorable for the fungus, nor as to resistant varieties. He mentions Fusarium as attacking the young growth, and speaks of bacteria associated with the rot of the bark and leaves. He did not find *Uromyces joffrini*, *Seuratia coffeicola*, nor *Seuratia vanillae*, which had been reported from Tahiti. The Capnodiaceae are mentioned.

Under the third chapter, which is on the control of the diseases, is discussed spraying. but the author appears to consider it impracticable.

Under chapter 4 on the Culture of the Vanilla, is described the amount of shade or sunlight desirable, the renewal of the plants, the selection and planting of the cuttings, nurseries, layering, distance of planting, care of the vines, and fertilizing of the flowers

Doubtless the object of the paper is attained in emphasizing the necessity of certain conditions for the growth of the plant. It is certain that this is of prime importance. It is, however, to be regretted that more specific information was not obtained as to the specific pests and diseases which attack this plant.

JOHN R. JOHNSTON

## PHYTOPATHOLOGICAL NOTES

*A seed-borne Sclerotium and its relation to a rice-seedling disease.* A seedling blight of rice that developed in the plats of the rice experiment station at Crowley, La., in 1914, was found by Dr. H. B. Humphrey to be due to an organism belonging to the *Mycelia sterilia* group of fungi. It was characterized by very small black sclerotia and a white sterile mycelium. In culture the fungus closely resembled the Sclerotium mentioned by Godfrey[1] as the cause of a leaf disease of rice, but no relationship between the two has been established. Later studies by the writer showed the seedling-disease fungus to be associated constantly with rice seed grown in Louisiana, particularly with the varieties Honduras and Blue Rose. Honduras rice grown in California, however, did not carry the fungus on the seed even though the original California sowings were made with Louisiana seed. In a single experiment, the fungus could not be isolated from Louisiana seed one year old, thus indicating that the fungus died during the course of the year. Seeds of several varieties from a lot of Chinese rice exhibited at the San Francisco Exposition in 1915 carried what appeared to be the same fungus.

The fungus under consideration was found not only upon the surface of the seed but embedded within and beneath the seed coat in the form of minute sclerotia. Seedling plants from surface-sterilized infected seed grown in sterile culture tubes quickly succumbed to the effects of the fungus, while plants from similarly treated non-infected seed would remain green for weeks. Clean plants grown in pure culture in tubes, inoculated with the fungus at the crown or roots, showed the same effects as those grown from naturally infected seed. Plants grown in pots and flats in the greenhouse were less susceptible to attack by the fungus than those grown in the laboratory. Evidently the greenhouse conditions were more favorable to plant growth and less favorable to the fungus, while in culture tubes the conditions were exactly the reverse. Such exact requirement on the part of the fungus for favorable conditions for development in order to do serious damage was repeatedly observed in various circumstances. However, many sowings of naturally infected and artificially inoculated seed in the greenhouse resulted either in the production of weak plants, from which reisolations were made, or in failure to produce plants at all.

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[1] Godfrey, G. H. Preliminary notes on an heretofore unreported leaf disease of rice. Phytopathology 6:97. 1916.

It was found that Jensen's modified hot-water treatment, namely, soaking in cold water for four hours and then treating with water at 54°C. for ten minutes, was capable of killing the fungus in hulled seed without materially injuring germination of the seed. This indicates the possibility of using some specially adapted modification of the hot-water treatment as a means of securing disease-free rice seed.

Owing to a change of duties the writer was obliged to discontinue further work on rice diseases. All notes on this, as well as on straighthead and other rice diseases, were turned over to his successor, Dr. W. H. Tisdale.

G. H. GODFREY.

*Errata.* Dr. F. D. Fromme calls attention to two important errors which appeared in the dates of dust applications in bulletin 223 of the Virginia Agricultural Experiment Station on "Dusting Experiments in Peach and Apple Orchards."

The date March 23, on page 5, should be April 23.

The date May 8 should be inserted in the Schedule of Applications on page 12.

Mr. F. V. Rand calls attention to two errors in the article on "Some Insect Relations to Bacillus tracheiphilus, Erw. Sm," published in PHYTOPATHOLOGY for March, 1920.

On page 133 the footnote reference in line 5 should be 3.

On page 138, in lines 7 and 36, December should be November.

*Personals.* Dr. Pier Andrea Saccardo, the distinguished mycologist and Professor Emeritus of the Royal University of Padua, Italy, died on the twelfth day of February, in the seventy-fourth year of his age. Professor Saccardo was a member of numerous academies and societies, both Italian and foreign, and is of course known to all pathologists and mycologists by his great Sylloge Fungorum, a compilation intended to include descriptions of all the published species of fungi of the world.

Dr. E. T. Bartholomew has resigned as assistant professor of Botany at the University of Wisconsin to accept a position as assistant professor in Plant Pathology, in the Graduate School of Tropical Agriculture and Citrus Experiment Station, Riverside, Calif.

# LITERATURE ON PLANT DISEASES[1]

COMPILED BY ALICE C. ATWOOD AND FLORENCE P. SMITH, BIBLIOGRAPHICAL ASSISTANTS, BUREAU OF PLANT INDUSTRY

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[1] This list includes references, both American and foreign, to the literature of plant pathology and mycology of interest to pathologists. Foreign references published since January 1, 1917, have been included beginning with the list appearing in v 7, no. 3, June, 1917.

All authors are urged to cooperate in making the list complete by sending their separates and by making corrections and additions, and especially by calling attention to meritorious articles outside of regular journals. Reprints or correspondence should be addressed to Miss E. R. Oberly, Librarian, Bureau of Plant Industry, U. S. Dept. Agric., Washington, D. C.

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# APPENDIX

## REPORT OF THE MEETING OF THE AMERICAN PHYTOPATHOLOGICAL SOCIETY, PACIFIC DIVISION, HELD AT PORTLAND, OREGON,

## APRIL 25 AND 26, 1919

The meetings were held at the Imperial Hotel following the Pine Blister Rust Conference for the West, held during the two days preceding at the same place. All of the meetings were well attended, the register showing forty names of persons from Ontario, British Columbia, Washington, Oregon, Idaho, Utah, and California, as well as a number of pathologists from Washington, D. C., and other parts of the country.

### AMERICAN PHYTOPATHOLOGICAL SOCIETY
### PACIFIC DIVISION

Friday and Saturday, April 25 and 26, 1919

### PROGRAM

Friday, April 25

*Morning Session*—9.30 a.m.

#### TREE DISEASES

Problems in forest pathology. Haven Metcalf.
Peridermium harknessii and Cronartium quercuum. Contribution II. E. P. Meinecke.
The oak fungus disease and orchard wood decay. W. T. Horne.
Round Table discussion of tree diseases. Informal notes from various members.
Some species of Uredinales with many aecial hosts. Ellsworth Bethel.

*Afternoon Session*—2.00 p.m.

#### DISEASES OF TRUCK CROPS

Some studies of Verticillium wilt of potatoes. M. B. McKay.
Notes on three cabbage leaf spots in the San Francisco Bay market gardens. Elizabeth H. Smith.
Observations on the bacterial blight of field and garden peas in Montana.—Read by title. H. M. Jennison.
Some fungi found in Utah soils. G. L. Zundel.
Open discussion on miscellaneous diseases of truck crops.

Saturday, April 26

*Morning Session*—9.30 a.m.

### CEREAL DISEASES

Roll Call.

Studies on the injurious effects of seed treatment on different varieties of wheat in Oregon. Jessie P. Rose

Some preliminary physiological studies on formaldehyde treated wheat. W. M. Atwood.

Limitations of formaldehyde as a fungicide for seed wheat. Anna M Hurd.

Seed injury from fungicides and saprophytes determined by the physical conditions of the seed coats. Anna M. Hurd.

The distribution of the two species of stinking smut of wheat in Oregon. Jessie P. Rose.

The distribution of the species of wheat and oat smut in Washington, Idaho, and Utah. G. L. Zundel.

The smut shower of Washington. G. L. Zundel.

The effectiveness of the Haskell method in the treatment of oats and wheat. F. D. Heald.

Some experiments with the covered smut of oats. F. D. Heald.

A preliminary note on the foot rot of cereals in western Washington. B. F. Dana.

The black chaff disease of wheat in Montana—Read by title. H. M. Jennison.

Open discussion on miscellaneous diseases of cereals.

Business meeting of the Society.

*Afternoon Session*—2.00 p.m.

The use of corrosive sublimate in treating seed potatoes. G. R. Hill.

Some experiments on the control of onion smut in Oregon. H. P. Barss.

Dusting versus spraying for disease control in the Hood River Valley. LeRoy Childs.

New dusting materials. R. E. Smith.

Blackheart of apricots. Helen L. Czarnecki.

Studies on the Sclerotinia of apricots. Edith H. Phillips.

Artificial infections of pear and apple blight through the leaves. F. D. Heald.

Blight resistance in Pyrus. F. C. Reimer.

Some results with disinfectants in blight control work. F. C. Reimer.

Pear blight control in the Rogue River Valley. C. C. Cate.

A new dormant spray for San Jose scale, blister mite, aphis eggs, etc. C. C. Cate.

Spraying for apple anthracnose. Arthur Frank.

Open discussion on miscellaneous fruit diseases and their control.

[PHYTOPATHOLOGY for May (**10**: 273–319, Plate XI) was issued June 1, 1920.]

PHYTOPATHOLOGY

VOLUME X NUMBER 7

JULY, 1920

# THE APPLE BLOTCH AND BITTER-ROT CANKERS

JOHN W. ROBERTS

The apple blotch canker caused by *Phyllosticta solitaria* E. and E. and the apple bitter-rot canker caused by *Glomerella cingulata* (Stoneman) Spaulding and von Schrenk have been discussed by various writers, but chiefly as sources of infection in connection with the diseases on the fruit.[1] The present paper is a discussion of the cankers as such, with little regard to their importance as infection sources.

## THE APPLE BLOTCH CANKER

As affecting the life of the tree, the blotch canker is not, as a general thing, entitled to very serious consideration. The lesions are small and persist for a relatively short time. They are not sufficiently numerous to cause much damage in the commercial orchard which receives several sprayings each year. In poorly cared-for orchards, especially in the southern portion of the Middle West, many twigs may be severely injured and even killed. In the case of such susceptible varieties as Northwestern Greening and Missouri Pippin, the trees themselves may become so thoroughly infested over a term of years that eventually they are killed.

The blotch canker first appears as a raised, black, blister-like spot on a twig or water-sprout of the current year's growth. It may become slightly sunken with the approach of autumn, but usually it continues somewhat raised above or level with the adjacent healthy tissues until the following summer and often throughout its life. As the twig or water-sprout matures the canker becomes lighter colored; and finally, in its second year, assumes a light tan color in the older or central portion with a dark border denoting the extent of the second year's growth.

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[1] For references to the literature dealing with these cankers see: Roberts, John W. Apple blotch and its control. U. S. Dept. of Agr. Bul. 543, 1917. The sources of apple bitter-rot infections. U. S Dept. of Agr. Bul. 684, 1918

Infection can take place only on the young branches of the current year's growth. Vigorously growing water-sprouts are especially susceptible. The writer has at different times attempted to inoculate older twigs, both by spraying them with water containing spores and by inserting spores beneath the bark. In no case have infections followed these attempts. On the other hand, the young green twigs of the current year when sprayed with water containing spores have invariably become infected. Inoculations through slits have been unsuccessful even in these twigs. The manner of infection has not been determined. It may be that the germ tube is able to penetrate the young and tender epidermis of the presence of stomata may be necessary for infection.

Though the spores germinate readily, subsequent development is very slow, six to eight weeks elapsing after inoculation before the cankers become visible. During the year in which infection takes place and during the earlier part of the following year, *Phyllosticta solitaria* is apparently actively parasitic. It increases the size of the infected area, and most of the cankers are free from other fungi. After this time, however, the fungus makes little if any further growth. Other fungi invade the cankered area and form their fruits on its surface.

During its first year, the fungus appears to make some growth during the dormant season of the host. By the first of May in the Ozarks the canker has expanded considerably and the fungus soon reaches the limit of its development.

Toward the end of the second season and especially in subsequent seasons one is apt to find the fruiting bodies of species of *Phoma* or other fungi abundant in the old canker, with a paucity or more often an entire lack of the pycnidia and spores of *Phyllosticta solitaria.* Apparently the tissues of the host have changed so that they are able to resist further invasion by the last named fungus. It is possible that the fungus is no longer able to absorb the necessary food materials, either because of the thickened walls of the host cells or because of the osmotic pressure changes within them. It is probable, moreover, that the more firmly compressed tissues of the host prevent or check the further intercellular development of the fungus. The hyphae, very slow growing even under the most favorable conditions, then would be cut off from proximity to young, thin-walled cells, which alone they seem able to parasitize. Under such conditions one would expect, as indeed does occur, the copious formation of fruiting bodies eventually followed by death. The hyphae are of comparatively large diameter and grow quite slowly, even in the tissues of very young twigs. A check in growth might easily cause their death by leaving them remote from the supply of nutrient materials.

Late in September, 1918, Ben Davis twigs of the current year infected with blotch were removed from the tree and used as material for sections. Appropriate lengths of these twigs were fixed in a mixture of picric acid, mercuric chloride and alcohol and demineralized in hydrofluoric acid. They were then dehydrated, embedded in celloidon, sectioned and stained. The most satisfactory stain used was the Pianeze IIIb as recommended by Vaughan.[2] This stain gave excellent differentiation and was in every way very satisfactory.

The hyphae within the tissues of the host are intercellular and of rather large diameter in proportion to their length. The tips are broad and blunt, often being broader than the main portion of the hyphal thread.

The infected cells are those of the cortical parenchyma, and the cambium. It is apparent therefore, that only thin-walled, not fully differentiated parenchymatous or meristematic tissues are invaded. The cells in the diseased area are filled with gum and are for this reason easily distinguished from cells remote from the infected area. Since only tissues composed of young, thin-walled cells are affected, the reason why only the young green growth of the current year is susceptible becomes apparent.

Pycnidia containing spores were found in the sections, some of them being located so far beneath the epidermis and so deeply embedded in the host tissues as to suggest that they may be blind pycnidia, *i.e.*, pycnidia which would not reach and rupture the epidermis.

In orchards that are regularly sprayed for the prevention of blotch infections on the fruit, the cankers gradually decrease in number until the trees are nearly free from them. The spraying prevents the formation of new ones, and the older ones, in which the fungus soon dies, heal over.

By coating the young twigs with the spray, infection by the fungus is largely prevented and hence few new cankers can be formed. Also, spraying, by preventing infections on the fruit, lessens in number the sources from which could arise spores capable of bringing about twig infections.

### THE APPLE BITTER-ROT CANKER

The direct damage to the tree by this canker is usually slight except in the case of unusually susceptible varieties, but beside its importance in connection with bitter-rot of the fruit, its non-occurrence in certain sections in which bitter-rot is prevalent and its peculiar host relations make it of interest to plant pathologists. The writer has discussed these phases of the subject somewhat in detail in a previous paper.[3]

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[2] Vaughan, R. E. A method for the differential staining of fungous and host cells. Annals Mo. Bot. Gard. 1: 241–242. 1914.

[3] Roberts, John W. The sources of apple bitter-rot infections. U. S. Dept. Agr. Bul. 684, 1918.

The canker begins by lodgment of the spores of the fungus in a wound or in a dead area. If conditions are favorable, *i.e.*, if moisture is present and the temperature is high, germination and subsequent infection take place very rapidly. When inoculations are made in mid-summer the canker becomes evident in a week or less. About the inoculated point, a rim of blackened bark appears, which may within a month attain a width of 50 mm. As the weather becomes cooler, growth ceases. In one series of inoculations there was no further growth after the first of August, the temperature being cool throughout the remainder of the season. Inoculations made in the autumn were unsuccessful.

As soon as increase in the size of the canker ceases, a layer of callus begins to form at the margins of the canker, which by mid-summer of the year following has rather well isolated the cankered portion of the bark. In this way the host usually prevents further growth of the canker, since the callous layer not only prevents spread of the initial canker but also effectively prevents the occurrence of new infections. The fungus lives and fruits in the isolated canker during the first and second years, but in the third year it was found to be dead and the lips of the callous layer nearly closed over the wound in nearly all the cankers artificially produced by the writer. In the case of especially susceptible varieties, such as Givens, the cankers may persist for many years. Givens does not start growth until very late in the season, often two weeks later than most varieties, and is peculiarly lacking in vigor. Cankers on the branches of Givens are often of large size and show the usual characteristics of the bitter-rot canker, *i.e.*, a blackened, depressed area with the bark clinging tenaciously to the wood. They also show concentric rings indicative of their age.

On June 28, one-year-old Ben Davis twigs were inoculated by introducing spores of the fungus into the bark through slits. By the first of August there was about the slit a blackened band 5 mm. wide, which after that time did not increase in size. By October the margin of the canker had begun to show slight rifts, indicating that the host had formed a callous layer about the dead area. On October 2, appropriate lengths of these cankered twigs were removed and after treatment with hydrofluoric acid and a mixture of glycerine and alcohol were sectioned without embedding. In all the sections the mycelium was found to be intercellular. In no case could a single filament be found within the cells. Hyphae were also found in the portions of the cankered bark which were adjacent to and sometimes clinging to the layer of callus, indicating that the growth of the fungus had kept pace with the death of the cells and that the cessation of development of the canker was not due to isolation of the fungus through the death of cells in advance of its growth. As before stated, cessation of growth was probably due to the lack of the high temperature necessary for the development of the fungus.

In the sections, even when viewed macroscopically, the most noticeable thing was the brown coloration of the diseased portion due to the formation of gum within the cells. In the cankered portion gum permeates the tissues at least as far as and often includes the pith. Even the cells of the outermost layer of the callus contain slight deposits of gum, which was probably formed originally within cells that by later division took part in the formation of callus. It is, of course, most abundant in the bark and cambium but it is also to be found in the wood and especially in the pith rays. So filled with gum are the last named that they appear as yellowish-brown bands running through the wood from pit to cambium. They are in sharp contrast with the bright, silvery wood, which is only flecked with the yellowish-brown gum, an effect due not to the absence of gum in the lumina but to the thickness of the cell walls and consequent relative smallness of the lumina. In the vessels the gum is bright yellow. It is doubtless the gum which causes the dead bark to adhere so tenaciously to the underlying tissues. The callus is the usual wound callus described by many investigators. It is composed of layers of thin-walled suberized cells and is able to isolate the canker, as the sections show, in a surprisingly short time. The number of layers composing the callus varies from six to twelve even in the same section.

A series of inoculation experiments made by the writer has indicated that there is considerable difference in the susceptibility of different varieties to infection. York Imperial proved to be practically immune while Yellow Newton was almost as resistant. Ben Davis, Jonathan, Missouri Pippin, Grimes Golden and Winesap were moderately susceptible but were not nearly so susceptible as Givens.

As before stated, bitter-rot cankers are usually rather short lived. Inoculation experiments show, however, that while the cankers on young, vigorous twigs or water-sprouts are usually healed over by the third year and are chiefly dangerous as infection sources during the second year, in the case of older, slow-growing branches there is not the same vigorous development of callus and the canker may therefore be relatively long-lived.

Bitter-rot cankers are most easily found during the dormant season when they are not concealed by foliage, or at the time when the first infections appear on the fruit. Cankers, when present, may often be found just above clumps of the earliest affected fruits. During and immediately after a series of years in which bitter-rot has been severe in orchards of the southern part of the Middle West, bitter-rot cankers are usually fairly abundant. Otherwise they are relatively scarce and hard to find. Usually the writer has found them most numerous on the variety Givens, but they are often abundant on Jonathan and Ben Davis.

BUREAU OF PLANT INDUSTRY
U. S. DEPARTMENT OF AGRICULTURE

# OBSERVATIONS ON THE DISTANCE OF SPREAD OF AECIOSPORES AND UREDINIOSPORES OF CRONARTIUM RIBICOLA

WALTER H. SNELL

As part of an intensive campaign to get definite data on several points in the life history of *Cronartium ribicola,* the writer undertook certain phases of the problem of the distance of spread of the aeciospores and urediniospores. The work was carried on under the general direction of Dr. Perley Spaulding, in charge of the scientific investigations on the white pine blister rust by the United States Department of Agriculture. The present paper deals with results obtained in the Adirondack Mountains, New York, in 1918, and near Rush Lake, Minnesota in 1919. These results were obtained by the employment of indirect methods. Spores were not caught in traps, but observations were made upon natural infections on pines and Ribes under the proper conditions. The investigations were undertaken with a view to obtaining data on the potential distance of spread of the spores. In the case of the aeciospores, some such evidence was obtained, but drought conditions prevailing at the time of the observations upon the urediniospores changed the aspect of the investigation from the potential distance of spread to that of behavior under dry conditions.

## DISTANCE OF SPREAD OF AECIOSPORES

East of Rush Lake in Minnesota in the spring of 1919, an area favorable for making the desired observations was located. Rush Lake is crescent-shaped with its axis east and west and the horns of the crescent to the south. Within the semicircle to the south of the Lake is a large scattered pine infestation, and to the southeast and east of this lies an area of several sections almost devoid of pine. It is level country, mostly cultivated farm land and meadow, with some hardwood woodlot and pasture, and some hardwood and tamarack swamp. In this area there were many "centers" of infection with no pines within one-half to one mile. In one large portion of this area a Ribes center was found on June 12, one and one-quarter miles from any pine whatever. The first uredinia were located in that area on June 6, and inasmuch as twelve to fifteen days is the incubation period for the uredinia of *Cronartium ribicola* under the most favorable conditions, any uredinia found within ten days after the finding of the first uredinia of the season were considered of the first generation. Daily search for uredinial infection was being made in this immediate vicinity by several scouts and there could, therefore, be no doubt but that the first uredinia were located about as soon as they appeared.

The inspection of the pines was done in the spring. In the fear that there might have been some more pines in the heavily leaved hardwoods or in the tamarack swamps then impenetrable because of abundance of water, another inspection of the area was made in November after the leaves were off the hardwoods and tamaracks, and when the swamps were frozen. Inspection made under these more favorable conditions revealed no pines within the radius of one and one-quarter miles. Hence, there is no doubt but that the aeciospores had to travel at least this one and one-quarter miles in order to infect this particular bush.

The weather conditions during the spring of 1919 near Rush Lake were very moist and hence very favorable for the spread of the rust, and prevailing westerly winds spread most of the infection east and southeast. The center used for distance determination was by no means the only one found. It was one of many infected bushes on the outer edge of the fan-shaped zone of infestation spreading to the east and southeast from the original pine center. At the distance of one to two miles, infected bushes could be quite readily found in the wood-lots. These facts are presented to show that the center chosen was not merely a single far-flown infection and therefore quite out of the ordinary, but was one of many in a zone more than a mile from the nearest pine and fully three miles from any pine at present known to be diseased. It is reasonable to assume that beyond this zone in which many infected bushes were found there should have been scattering infections for some little distance. In fact, some infections were found up to the 14th day following the finding of the first uredinia of the season, but it was deemed proper to include as definite first generation infections only those found within 10 days.

Observations made near the villages of Essex and Whallonsburg in Essex County, New York, in 1918, gave similar results. The most promising situations were surveyed, and on June 15, before the second generation of urediniospores appeared, many first generation centers were located near the central points of the larger areas. It was a simple matter to establish aecium-free areas with a quarter mile radius, that is, centers consisting of first generation uredinia one-quarter of a mile from any pine whatsoever. Little attention was paid to these, however, as longer distances were sought. Center J had no pines whatever within 1240 yards—nearly three-quarters of a mile. The center consisted of but one uredinial spot upon a *Ribes cynosbati* bush on the edge of a small swamp surrounded by meadowland, more than half a mile from any road. Centers E and F had no pines at all within 800 yards and only one within 1100 yards. This pine was a small one fifteen feet tall. Close inspection revealed no blister rust, and several *R. cynosbati* bushes in the fence-row nearby not only had no disease at the time of inspection, but remained healthy all summer. Center A likewise had a small pine 15 to 20 feet tall within a half-mile radius. The pine grew by the side of a road and had been trimmed so that there was not a great amount of foliage. It was easily climbed and inspected minutely, and found to be free of infection. Three *R. cynosbati* bushes directly under it were uninfected at that time and remained so all summer. With reference to centers E, F and A, it is probable that there were no aecial infections within the maximum distance given.

In considering the above cases it may be assumed that the wind carried the aeciospores through the air at least one and one-fourth miles in one instance, although it may be objected that the spores might have been brought by other agencies, such as vehicles, dogs, horses, cattle, etc. In the Minnesota study, such possibilities are very remote. There were too many infected bushes over too wide an area to be attributed to such agencies, especially when there is a heavily infected pine lot in the vicinity. In the New York area, the three-quarters-mile center was more than half a mile across hay meadows, where neither vehicle nor animal transmission need be considered.

Further objections may arise with reference to the possible overwintering of spores of *Cronartium ribicola*, which might start infection of the Ribes at distant points from diseased pine. The possibility of *C. ribicola* spores retaining their viability through the winter has been demonstrated by Miss Dosdall (1) in the case of aeciospores, and by York and Spaulding (2) and Miss Taylor (3) for urediniospores, but thus far, there is no case on record in which it has been proved that overwintering spores have started natural infections on Ribes. And furthermore, while it is possible that isolated infections might arise from overwintering spores, no one has yet maintained that overwintering could be responsible for infections on such a scale as at Whallonsburg in New York, where 25 per cent of the bushes over ten or twelve square miles were diseased, to say nothing of the much more intense infection at Rush Lake in Minnesota.

## SPREAD OF UREDINIOSPORES DURING A DRY SUMMER

Late in June, 1918, in Essex County, New York, just prior to the appearance of the third generation of urediniospores, the relatively slight degree of infection of the Ribes bushes in an area where the pines were generally affected was so pronounced as to invite further attention. It was expected that by the last of June the third generation would have appeared, accompanied by considerable dissemination of the disease. What was actually found, however, was that the third generation appeared some days later than was expected and that it had not sprung up either over a wider area or much more heavily within the area restricted by the bounds of the primary uredinial infection. Infections were found to be confined to the same bushes noted as infected earlier in the season and not to have spread to nearby bushes, and said infections were still confined to portions of the bushes within a small radius of the primary infections. Some bushes, of course, were entirely infected, but it was evident that they had been heavily and generally infected by aeciospores at first. Other bushes showed but one isolated branch infected, perhaps one extending out from the body of the bush. Still others showed localized infections of spherical or conical shapes, with the first generation of uredinia at the center of a group of leaves or at the apex of a cone of infected leaves.

Two plots, one at Lewis, and another at Whallonsburg, New York, were put under observation. The plot at Lewis consisted of ninety-nine bushes of *Ribes cynosbasti* and *R. rotundifolium* beneath and around two infected pines to a distance of about 100 yards, partly in medium aged pine growth and partly in open pasture. The plot at Whallonsburg included about seven square miles and was for the most part open country under cultivation, with very little land as woodlot. There was one lot of cultivated red currants. The wild Ribes were few in number and found scattered in woodlots and pastures, but for the most part along fence rows. The species were *R. cynosbati*, *R. rotundifolium* and *R. americanum*. In all, there were 102 bushes under observation. The individual bushes were tagged and numbered, and notes made on the condition of each bush, whether infected or not, the amount and extent of infection, and whether the infections were localized and restricted or general over the bush. Observations were made and notes taken thereafter every two weeks until the end of the season.[1] As the results were much the same for the two plots, even as to percentage of infections, etc., they will be discussed together and discrepancies will be pointed out. The more salient facts are presented in table 1.

Of the 201 bushes in both plots, 111 were infected by the aeciospores in the spring as shown by the number of generations of uredinia on the leaves at the time of the first observations. Of the ninety bushes not diseased in the spring, only nine (eight at Lewis and one doubtful case at Whallonsburg) contracted any disease during the summer. The remaining eighty-one were still disease-free in the fall. Six of the eight bushes at Lewis were very slightly infected, having only two or three leaves diseased. Two bushes were moderately infected. In the case of these eight bushes at Lewis there is the possibility of dissemination by horses and cattle pastured in the lot, or by berry-pickers. Eight bushes (five at Lewis and three at Whallonsburg) found diseased in July were not diseased in September, probably because the infected leaves had fallen off.

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[1] The final set of observations were made by Dr. L. H. Pennington and Mr. D. M. Benedict, because of the premature closing of the writer's work in that section.

There was very little dissemination from diseased to healthy bushes during the summer, and the spread within the originally infected bushes was slow and restricted in most cases. That is, the spread was what might be termed localized, in contradistinction to a general spread. In fourteen of the bushes at Lewis, the spread was general; the leaves became infected all over the bush so that there were no well-defined centers or groups of diseased leaves. In the centers or groups of leaves, more or less spherical or conical in shape (cf. "infection cone" in case of bitter-rot of apple (4), p. 37, (5), p. 12, and (6), p. 576), the original dried-up lesions with browned and dried uredinia could usually be made out, surrounded by lesions with their uredinia in various stages, from old to fresh, bright-colored ones. Such centers or groups of infected leaves appeared as if the whole had originated from one or more uredinial spots, presumably from aeciospore infection, and that then each succeeding generation of uredinia had sprung up quite locally and within a very short distance of the preceding sporulating uredinia. At the time of the third generation of uredinia, the case was very plain. The new uredinial spots and uredinia were bright and fresh (third generation), there were a number of older uredinia, brown and faded in color with the spots drying up (second generation), and in the midst of all these, one dried leaf or more bearing old darkened uredinia (the first generation). Such local infections—using "infections" here

TABLE 1

*Combined results of the observations on dissemination of the urediniospores of Cronartium ribicola during the dry summer of 1918 at Lewis and Whallonsburg, New York*

| | TOTAL NUMBER OF BUSHES | NUMBER INFECTED IN SPRING | NUMBER INFECTED IN FALL | SPREAD LOCAL BY FALL | NO SPREAD BY FALL | SPREAD GENERAL BY FALL |
|---|---|---|---|---|---|---|
| Lewis .......... | 99 | 50 | 62* | 45 | 0 | 14 |
| Whallonsburg.......... | 102 | 52 | 50* | 43 | 9 | 0 |
| Totals........ | 201 | 111 | 112 | 88 | 9 | 14 |

* Eight bushes at Lewis uninfected in the spring contracted the rust during the summer, but five of those originally infected in the spring lost their infected leaves and were not diseased in the fall. At Whallonsburg, one new bush became diseased, but three of those diseased in the spring were not diseased in the fall.

to signify these definable groups of infected leaves—were observed during the course of the summer, and it was evident that they preserved their individuality for the most part until late in August, increasing a little in size with each generation until all the leaves on the bush were attacked. As mentioned, fourteen bushes at Lewis were infected generally early in the season. Nine of those at Whallonsburg did not develop any more disease than was evident in the spring. But the remaining eighty-eight all gave evidence, both at the time of the third generation in early July and during the remainder of the summer, of this restricted, localized uredinial dissemination.

Intensity of the disease within the different bushes is a different matter, for dissemination might be general and yet not be very intense, or localized and yet heavy. The point to be brought out is that in about 70 per cent of the cases (70 in 102) where any increase in amount of disease was evident during the season, the rust, though spreading slowly and quite locally, proceeded to the point of defoliating the bushes.

From a similarity of results obtained from the two quite different plots, it appeared as if the dissemination of the blister rust on the Ribes was quite materially checked in this locality in 1918. One would naturally turn to the weather conditions for an explanation. The summer of 1918 was a dry one in Essex County, New York, particularly around Lewis where it is very sandy. Up to the middle of July there was an abundance of rain, but from July 17 to September 3 it was hot and dry. There were but few showers and there were four stretches of four to seven days without any rain at all. There were but three generations of uredinia between the first of July and the middle of September, the third about July 20, the fourth about August 19, and the fifth about September 12. The month of September was one of rain and high humidity. During this period the generations of uredinia could not be so easily distinguished because there was plenty of moisture to induce the formation of uredinia at any time. Much of the season's increase of disease upon the Ribes occurred at this time. It appears, therefore, that dry weather is likely to prevent general dissemination of the white pine blister rust by urediniospores.

## SUMMARY

Observations show that the aeciospores of *C. ribicola* can be blown more than one and one-fourth miles and infect Ribes. One center noted was one and one-quarter miles from any pine whatsoever.

Dry weather prevented a wide distribution of the disease by urediniospores during the sumer of 1918 in Essex County, New York.

OFFICE OF INVESTIGATIONS IN FOREST PATHOLOGY
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WASHINGTON, D. C.

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# HEAT INJURY TO BEANS IN COLORADO

H. G. MacMillan and L. P. Byars

With One Figure in the Text

In Colorado the month of June, 1919, was marked by quite extreme conditions of temperature. The month began with a record-breaking cold snap. At Greeley the thermometer fell to 29° F. on the second, and 28° F. on the third. At the end of the month high temperatures were experienced for a few days. The thermometer rose to 102° F. on the 28th, and 104° F. on the 29th. For the month, as a whole, the temperature averaged about normal.

These extreme conditions were evident in the appearance of the vegetables. The early planted crops were quite badly frosted, especially those in the seedling stage. Beans are usually planted about the middle of May or soon thereafter, and were in the tender, seedling stage on the second of June when the frost came. This killing frost made necessary the reseeding of considerable areas, which was done as soon as seed could be procured and planted. The planting of the second crop was done between the fifth and twelfth of June.

At the end of the month the second-crop beans were in the seedling stage, just acquiring the first two leaves or bringing them to full size. In fields on sandy land a very serious condition was noted, not readily accounted for at the time. The stems were shrunken at the ground line, the upper part falling or leaning over so that the leaves touched the ground. Many of the leaves were not noticeably wilted, although the first appearance of the plant suggested wilt from drought. Macroscopic examination revealed the underground stem and roots to be in normal condition. The stems were later examined for evidence of a damping-off fungus or other parasitic organisms, but no such organisms were found upon the seedling plants. The plants were not suffering from lack of water.

The plants shown in figure 1 exhibit typical symptoms of the malady. The stem had collapsed at the point just at or below the surface of the ground, and the tissues were shrunken and collapsed, though not dry. The collapsed tissues were not materially different in color from the turgid parts of the stem, and usually the epidermis remained unbroken. As no organism appears concerned in the attack, and because of the high temperatures, especially at the surface of the soil, prevailing at the time the injury first became apparent, it is presumed that the trouble was caused entirely by excessive heat. No records are available which have a direct bearing on the conditions in fields where this trouble occurred. However,

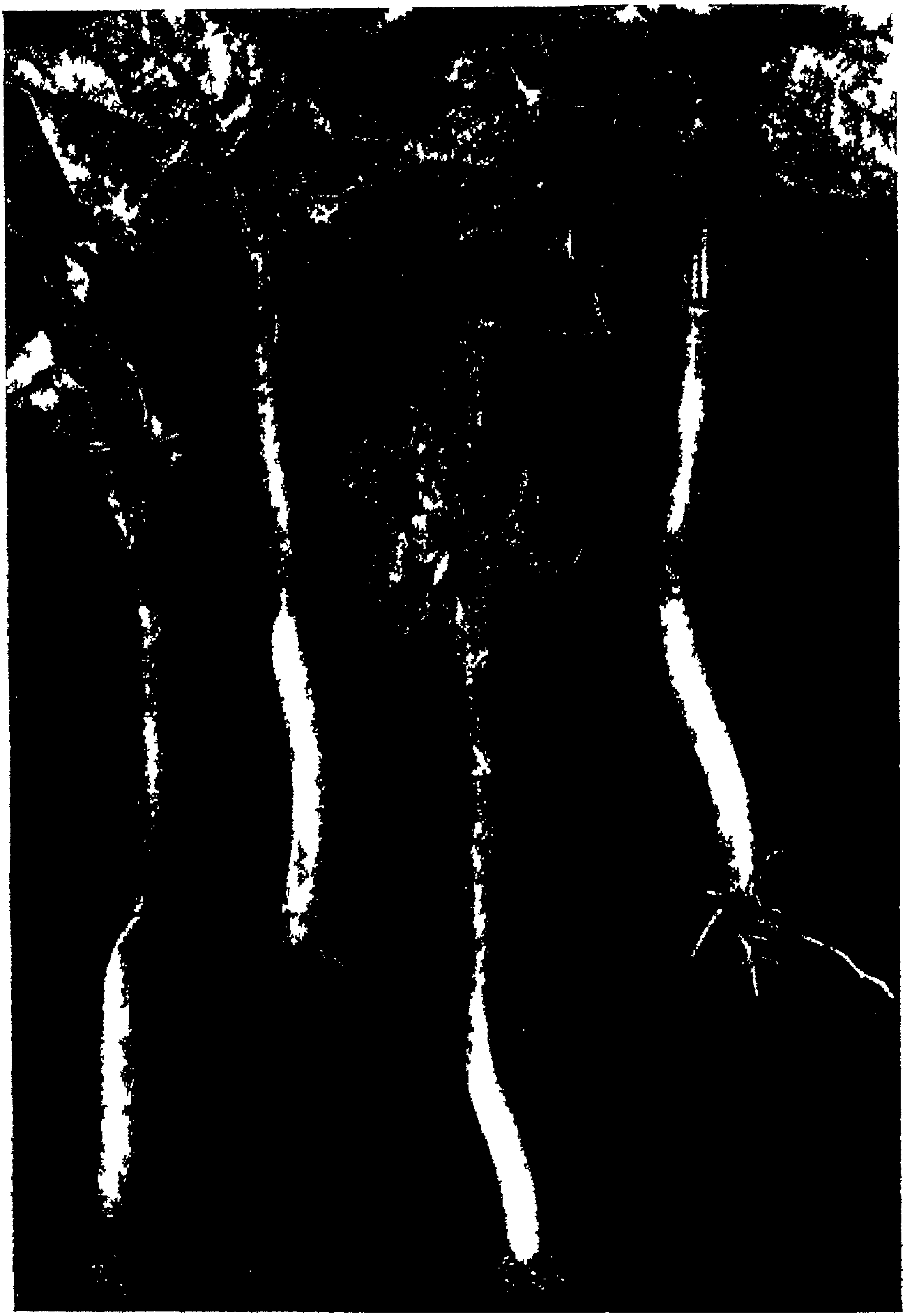

FIG. 1. BEAN SEEDLINGS AFFECTED BY HEAT INJURY

Note the slightly shrunken stems and turgid leaves of the upper seedlings which are not seriously affected and the seriously shrunken stems and wilted leaves of the lower seedlings which are badly injured. Plants collected June 30, 1919, from a sandy field near Greeley, Colorado, and photographed natural size.

a few miles away, at the Colorado Potato Experiment Station, soil temperatures were being taken in a potato experimental plot. The records were taken at depths of one and three inches below the surface, but none at the surface of the ground.

At a depth of 1 inch the temperature averaged 109.8° F. for the last ten days of June, rising to 112° on the 27th and 28th, and 113° on the 29th and 30th. At the same time, the ten-day average for the 3-inch depth was 92.3° F., the temperature reaching 94° F on the 27th and 28th, 93° F. on the 29th, and 95° F. on the 30th. The air temperature averaged 99.3° F. for the last ten days of June, rising to 104° F. on the 29th and 101° F. on the 30th. For the few days succeeding, the temperature was lower.

The soil temperatures given are only indicative. The soil upon which the beans were growing was quite light and sandy, and very hot. The soil at the Station was a Billings loam, a medium heavy soil, not becoming smooth on the surface under the influence of rain or irrigation water, as the sand would do. A year or more earlier at the Station some soil surface observations were taken with a mercury thermometer shaded from the sun. It was found then that it rose readily to 125° and 130° F. No doubt the beans were exposed to a temperature of 130° F. or higher for two or three hours each day, resulting in the heat injury. The plants which had suffered to the extent of being prostrated did not survive.

Hartley's[1] observations on stem lesions of seedlings of conifers, cowpeas, and rye, which he thought to be due to excessive heat, are of special interest in connection with the observations just presented. He found seedlings being killed both in the field and in poorly shaded nurseries, and mainly in Nebraska. The stems were collapsed and shrunken just at or a little above the surface of the soil, and in addition there was usually formed at the same place a definitely delimited lesion, which generally became white. On account of its color he described the trouble as "white spot."

The writers have not observed this characteristic "white spot" on the stems of beans in Colorado. Possibly it might have developed had higher temperatures prevailed or had other factors, such as light and moisture, been identical with those in Nebraska.

It has seemed desirable to present the above observations unsupported by experimental data, because opportunity may not permit of carrying out further investigations on the subject by the writers. Furthermore, it is hoped that this note may add something to the small amount of data available on the pathological importance of high soil temperature.

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[1] Hartley, Carl. Stem lesions caused by excessive heat. Jour. Agr. Research **14**: 595–604, fig. 1. 1918.

# BRIEFER ARTICLES

## NOTE ON RESISTANCE OF CHESTNUT TO THE BLIGHT

J. FRANKLIN COLLINS

WITH TWO FIGURES IN THE TEXT

Ever since the writer first became actively interested in the chestnut blight (*Endothia parasitica* (Anders.)Murrill) in 1908, he has been on the lookout for suggestions of immunity or resistance in individual plants of various species of the genus *Castanea*. Many sprouts and trunks of trees that appeared to be resisting the disease, or that were suggesting immunity at the time of observation, have been noted. In nearly all of these the period of resistance or apparent immunity proved to be of comparatively short duration, though in a few cases the period extended over two or three years; rarely longer. In a great majority of such cases the apparent immunity was probably due to the uninjured condition of the bark, while temporary resistance could in some cases be rather definitely traced to an unusually vigorous growth of the host, due to particularly favorable growth conditions. Disappointing experiences of this sort were duplicated so many times between 1908 and 1913 that the writer became very reluctant to make any definite statement in reply to inquiries regarding the prospects of locating immune or resistant chestnuts other than the Japanese, to which Metcalf first called attention in 1908.[1] Obviously, no reliable final statement regarding such conditions can be made until observations have extended over quite a number of years.

However, down to the present time the writer has been constantly on the lookout for trees that showed suggestions of immunity or resistance. Hundreds of trees have been under observation during the last eleven years, and at the present time numerous trees are still under periodical observation. In general it may be stated that resistance has been more pronounced in the last few years (four or five) than in earlier years. This last statement is meant particularly to imply that in many cases observations over a greater number of years are now necessary to disprove the suspicion of immunity or resistance than was the case formerly; in other words, this apparently means that (a) the disease is progressing more slowly, or (b) the host is more successfully resisting the disease, or (c) the more susceptible trees have all been killed, or (d) some other factors, are influencing results.

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[1] Bureau of Plant Industry Bulletin 121, part 6, pp. 3-4. February 10, 1908.

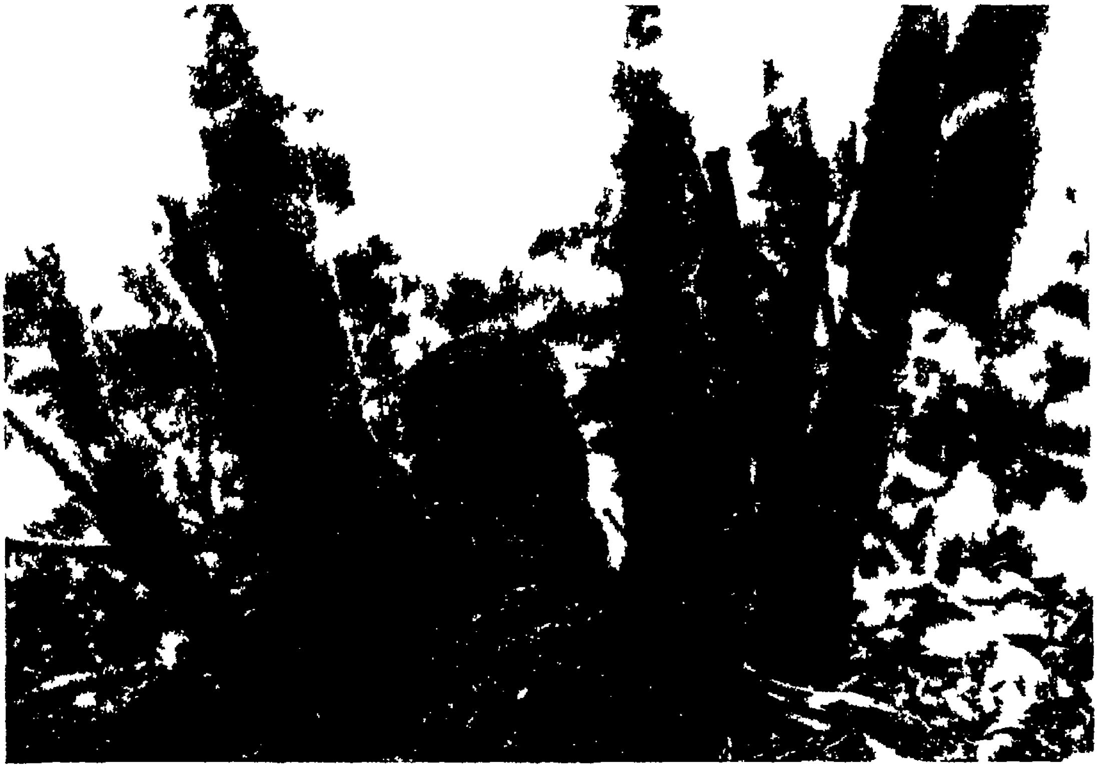

Fig 1 From Photograph Taken November 13 1912

*A* The living diseased sprout particularly referred to in this article *B* Stump from which sprouts originated *C* Living sprout diseased at base *D* Small dead forked branch of sprout *E* *E* Badly diseased and practically dead sprout

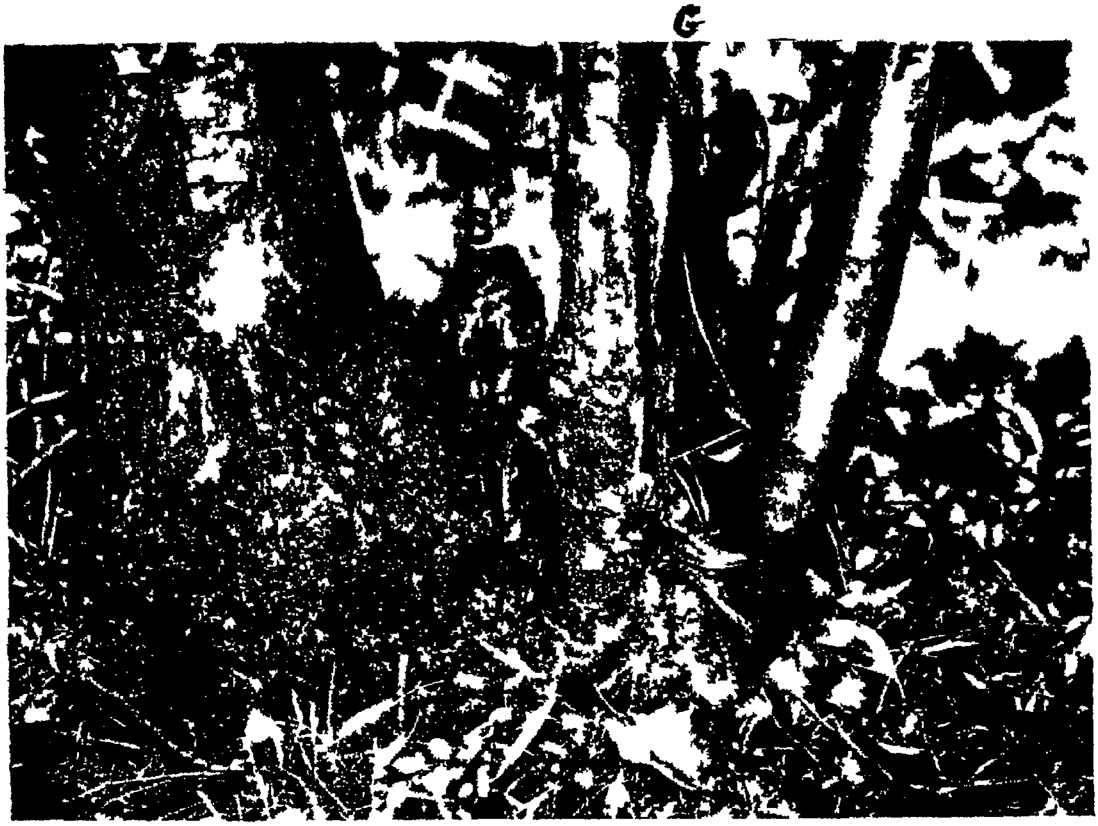

Fig 2 Photograph taken July 13 1917 (same distance from sprouts as in fig 1)

Letters refer to the same sprouts as in figure 1 *C* Sprout apparently dead since 1913 *F* New living sprout directly in front of *E* *G* New living sprout behind *C* and *D*

Among the many sprouts of American chestnut trees that have come under the writer's observation, from the point of view of possible resistance, the case mentioned below seems particularly worthy of note at this time.[2]

On November 13, 1912, the writer took a photograph of some chestnut sprouts in Lancaster County, Pennsylvania, at least one of which showed a well marked, characteristic, though not an extreme, case of disease, with accompanying hypertrophy and prominent coloring at the base of the stem (fig. 1). These sprouts had evidently grown from the stump of a small tree cut three or more years earlier when the undergrowth surrounding some grafted orchard trees was mowed. The exact age of the sprouts was not determined.[3]

At the time the photograph was taken the largest living sprout was approximately 3¾ inches in girth just above the hypertrophy, and presented the same general diseased appearance as hundreds of other sprouts that died one or two years later. Much to our surprise, this sprout continued to live and grow, the hypertrophy at the base becoming less and less pronounced in subsequent years, until in 1917 the swelling was scarcely apparent, as will be seen from the accompanying illustration (fig. 2).

On the date of the latest records (September 24, 1919), the main sprout was 16 feet high and measured 1 foot 3¾ inches in girth, 1 foot from the ground. The old hypertrophy of seven years earlier was no longer in evidence, except perhaps as a rougher surface to the bark at the base. There was some suggestion of the yellow color so often accompanying the disease, in the cracks at the base, but no sign of even a faintly delimited lesion or hypertrophy. Formerly there was a disease canker two feet from the ground. This was nearly healed over in 1919. The callus surrounding it showed some old pustules, but no other external indication of active disease. Another canker ten inches long showed little evidence of disease in 1919.

Although this sprout still is diseased in several places and several twigs have been killed, there are at least three places on the main stem where disease formerly was present, but now, from external examination only, is apparently absent or very nearly so; moreover, the foliage of the crown appears to be essentially normal. No cutting to ascertain the condition of the inner bark has been made at any time.

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[2] For resistance in mature trees see Graves, Arthur Harmount. Resistance in the American chestnut to the bark disease. Science n.s. 48: 652–653. December 27, 1918.

[3] The small stump from which these sprouts grew was 4 or 5 inches high and approximately 2½ inches in diameter on the date mentioned above. At the time of the first recorded observations on this plot no tree was indicated at this point, so the cutting undoubtedly was done prior to 1909. Like numerous other trees nearby, this may have been an American seedling tree grafted with a Japanese scion, which later was cut below the graft; but there was no evidence to prove this. The existing sprouts are of the ordinary American chestnut, so far as can be judged from foliage specimens only.

Between the years 1912 and 1917, the sprout increased in girth from 3¾ inches to 13½ inches, an average yearly increase of nearly 2 inches. In the subsequent two years, the average yearly increase was 1½ inches.

Of course, no prophecy can be made as to the probability of the sprout finally entirely overcoming the disease, but the fact that any badly diseased sprout, but little over an inch in diameter, should survive seven years and increase to a diameter of approximately five inches in that time seems specially worthy of note.

Perhaps some reader may recall instances of resistance that have been observed; if so, the writer would be glad to hear of them.

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## NOTE ON THE PATHOLOGICAL EFFECTS OF BLAZING TREES

JAMES R. WEIR

Wounds in trees susceptible to infection serve cull-producing fungi in two ways. A fungus already in the heartwood is enabled to penetrate the exposed tissues and produce sporophores in exactly the same manner that sporophores are produced from branch knots and injuries caused by fire and weather; secondly, wounds are a source of infection from without.

The danger of infecting conifers through mechanical wounding, such as blazes, logging scars, and various injuries to the sapwood attending general forest improvement work, largely depends upon the quantity of resin secreted from the exposed surface. For the present purposes the conifers of this region are classified as resinous and non-resinous species. This classification is based entirely on whether or not a tree when blazed secretes a protective coat of resin. The usual anatomical classification is disregarded. A wound in the sapwood of a resinous species is almost immediately protected from infection by a heavy exudation of resin, which is not the case in the latter group. With this observation as a basis, a large number of blazes during the past seven years have been examined in Montana and Idaho, to determine their relation to the entrance or exit of cull-producing fungi, as evidenced by the presence of sporophores on the exposed wood, special attention being given to blazes because they are preventable.

Results of this study are embodied in table 1.

The sporophores found on the blazes were of the following species: *Trametes pini*, *Fomes pinicola*, *Fomes laricis*, *Hydnum* sp. (unnamed), *Echinodontium tinctorium*, and *Pholiota adiposa*, and are the species which are responsible for most of the cull in the trunks of coniferous trees in the region studied. *Trametes pini* is more or less common to all conifers, as is *Fomes pinicola*, which is frequently a wound fungus. *Fomes laricis* is chiefly associated with larch in the region studied. The last three named species are practically confined to hemlocks and firs.

The foregoing shows quite clearly the rôle blazes play in the development of sporophores, also that blazes in non-resinous conifers are a more prolific source of sporophore production than in resinous species. The table shows

TABLE 1

*Number and species of conifers examined, number of wounds on each class, and number of wounds bearing sporophores for each class*

| | NUMBER OF WOUNDS BEARING SPOROPHORES | | | | | | NUMBER OF WOUNDS EXAMINED | | | | | | NUMBER OF TREES EXAMINED | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | WP* | L | DF | LPP | YP | Total | WP | L | DF | LPP | YP | Total | WP | L | DF | LPP | YP | Total |
| Resinous trees | 2 | 2 | 3 | 2 | 1 | 10 | 13 | 12 | 10 | 12 | 7 | 54 | 25 | 32 | 21 | 12 | 9 | 99 |
| | AF | ES | GF | WH | | | AF | ES | GF | WH | | | AF | ES | GF | WH | | |
| Non-resinous trees | 20 | 10 | 25 | 8 | | 63 | 15 | 10 | 21 | 4 | | 50 | 10 | 23 | 25 | 20 | | 78 |
| Total .. .. .. ... .. | | | | | | 73 | | | | | | 104 | | | | | | 177 |

*WP = Western white pine.
L = Western larch.
DF = Douglas fir.
LPP = Lodgepole pine.
YP = Western yellow pine.

AF = Alpine fir.
ES = Engelmann spruce.
GF = Grand fir.
WH = Western hemlock.

86 per cent more sporophores on wounds of conifers listed as non-resinous species than on resinous species. Though anatomically belonging to the resinous group, Engelmann spruce is here listed with the non-resinous species, owing to the fact that blazes on this tree do not secrete resin as readily or in as large a quantity as is the case in pines, and that such wounds more frequently bore sporophores than was the case with the trees listed as resinous. These sporophores may have developed from mycelia already in the heartwood of the trees, a condition to be expected in a study of this kind. The spruce in the regions chiefly studied was very defective. The wood of spruce, however, is by no means as resinous as that of the pines.

The danger from blazes may be largely eliminated by making light blazes in the bark and bast which will, in most cases, be quite sufficient for the purpose in hand, and are not readily susceptible to infection. This is particularly true in the case of thick bark trees. By careful manipulation of the ax a blaze in any tree may be made in the bark without injury to the wood, and spores which may be carried on the ax will not find a suitable substratum. The practice of using tags, as in the case of marking trees on timber sale operations, is an excellent plan. Since they are put on with tacks or small nails and would come off in the slab on sawing, and do not afford an entrance to fungi, their use should be encouraged.

## SUMMARY

On the basis of resin secretion from blazes, conifers are classified as resinous and non-resinous. An examination of a number of blazes from each class shows that they play an important part in the development of sporophores of cull-producing fungi, and that blazes in non-resinous species are a more prolific source of sporophore production than in resinous species.

OFFICE OF INVESTIGATIONS IN FOREST PATHOLOGY
BUREAU OF PLANT INDUSTRY
SPOKANE, WASHINGTON

# ALBERT THOMAS BELL

C. W. EDGERTON

WITH PORTRAIT

Albert Thomas Bell was born at Biggsville, Illinois, October 6, 1866, and died at Baton Rouge, Louisiana, July 27, 1919. When he was fifteen years of age, his parents moved to Furnas County, Nebraska, and a year later he entered the Lincoln (Nebraska) High School. He was unable to finish his preparatory training, and while less than eighteen years of age he began teaching in the rural schools of Furnas County. After teaching in the rural schools for five years and in the Crete (Nebraska) High School for seven years, he entered the University of Nebraska, from which he received his B.S. degree in 1898 and his A.M. degree in 1900. He specialized in botany and was made a member of Sigma Xi at that institution. After leaving the University, he taught in the Lincoln (Nebraska)

High School for two years and in the Nebraska Wesleyan University for eight years. For the last nine years of his life, he was professor of botany in the University of Louisiana.

Professor Bell was an exceptional teacher and was deeply interested in his chosen profession. The late Prof. Chas. E. Bessey, under whom he did his university work, once stated to the writer that he considered Professor Bell one of the best teachers that had ever been in his department. He spent his whole life in teaching and it can be truthfully said that he died in harness as he had a stroke of apoplexy while on a field trip with some of his students, dying within less than forty-eight hours without regaining consciousness.

Professor Bell was married in 1892 to Miss Belle Cooper, who still survives him.

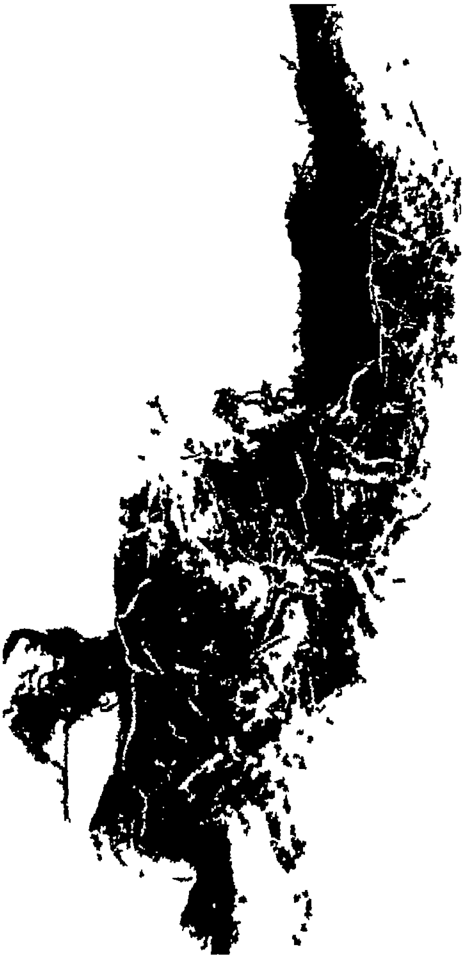

Fig. 1. Root of an Azalea Bush Killed by Shoe-String Root Rot

*Shoe-string root rot of Rhododendron and Azalea caused by Armillaria mellea Vahl.* *Armillaria mellea* has been reported as causing important root rots of various kinds of coniferous trees and deciduous trees and shrubs. It is the purpose of the present note to report an extreme case of root rot of Rhododendron and Azalea caused by this fungus in the Northwest.

A group of Rhododendrons and Azaleas in a large bed of these shrubs was noticed to die during the spring of 1915. On being examined it was found that the root systems were almost completely enveloped by rhizomorphs of *Armillaria mellea*. Little attention was paid to the matter until the following spring when it was observed that more of the shrubs had been killed. The infected area is still gradually enlarging.

The general appearance and the extent of the infection on an Azalea root system is shown in figure 1.

Henry Schmitz

*Personals.* Mr. L. O. Watson has resigned his position as pathological adviser in the Office of Cotton, Truck and Forage Crop Disease Investigations, Department of Agriculture, to engage in a private seed-growing business at Florence, S. Car.

Mr. Geo. M. Armstrong, recently a graduate student at Washington University, St. Louis, has accepted the position of assistant pathologist in the Bureau of Plant Industry and has taken up work on the control of the root-knot nematode.

Mr. Freeman Weiss, formerly a joint employee of the Department of Agriculture and the Minnesota Agricultural Experiment Station, engaged in the investigation of cereal diseases, has accepted a full time appointment as assistant pathologist in the Department of Agriculture and is engaged in the investigation of the potato wart disease.

Mr. N. Rex Hunt of the Office of Forest Pathology Investigations, Bureau of Plant Industry, has been transferred to the Federal Horticultural Board to assist in the eradication of the potato wart disease.

Dr. Charles Drechsler has been transferred from the Office of Cereal Investigations, Bureau of Plant Industry, to the Office of Cotton, Truck and Forage Crop Disease Investigations and has taken up the investigation of vegetable decays and decay-producing organisms, with headquarters at Brooklyn, New York.

Mr. D. C. Neal has resigned the position of plant pathologist for the Georgia State Board of Entomology to accept a similar position at the Mississippi Agricultural Experiment Station.

Mr. C. M. Tucker, a recent graduate of the University of Missouri, has been employed by the Extension Division of the Florida College of Agriculture to conduct extension work on the control of watermelon diseases.

Dr. R. O. Cromwell has resigned from the Office of Cereal Investigations, U. S. Department of Agriculture, in order to accept a position as crop specialist with the firm of E. W. Wagner & Co., Chicago, Ill.

Dr. W. H. Weston returned to Washington, D. C., on April 29. Since December, 1917, he has been in the Orient, principally the Philippines, studying the downy mildew of corn.

Dr. F. Kolpin Ravn, of Denmark, visited Washington in May, and addressed the Botanical Society of Washington May 4, on Plant Pathology in Denmark in recent years. We record, with deepest regret, the sudden death of Dr. Ravn, from blood poisoning, on May 25, at the home of his wife's parents, at East Orange, New Jersey.

# LITERATURE ON PLANT DISEASES[1]

COMPILED BY ALICE C. ATWOOD AND FLORENCE P. SMITH, BIBLIOGRAPHICAL ASSISTANTS, BUREAU OF PLANT INDUSTRY

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All authors are urged to cooperate in making the list complete by sending their separates and by making corrections and additions, and especially by calling attention to meritorious articles published outside of regular journals. Reprints or correspondence should be addressed to Miss E. R Oberly, Librarian, Bureau of Plant Industry, U. S. Dept of Agric, Washington, D. C.

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## SOUTHERN DIVISION

### MEETING OF THE SOUTHERN DIVISION OF THE AMERICAN PHYTOPATHOLOGICAL SOCIETY

The second annual meeting of the Southern Division of the American Phytopathological Society was held at Atlanta, Georgia, February 24 and 25, 1920, in conjunction with the meeting of the Southern Agricultural Workers. Pathologists from most of the southern states were present and took part in the round table discussions. C. W. Edgerton, president of the Southern Division, presided during the discussions.

The meeting of the first day was a joint session with the Southern Horticulturists and the program was limited to papers and discussions on sweet potato problems. The program of the second day consisted of discussions on some of the phytopathological problems which are receiving most attention in the Southern States. The program follows:

*February 24. Sweet potato problems*

| | |
|---|---|
| Fertilizers | H. W. Barre |
| Influence of time of picking on keeping quality | G. C. Starcher |
| Varieties | G. C. Starcher |
| Certification | J. A. Elliott |
| Transportation and storage | R. H. Hill |

*February 25. General discussions*

| | |
|---|---|
| Tomato wilt and resistant varieties | J. A. McClintock, C. W. Edgerton |
| Corn root and ear rots | H. R. Rosen, S. H. Essary |
| Truck crop diseases | C. D. Sherbakoff, W. C. Herron |
| Mosaic diseases, with special reference to a sweet potato mosaic | H. R. Rosen |

After the program, the following officers were elected for the ensuing year.

*President*, S. H. Essary, Tennessee Experiment Station.

*Vice president*, J. A. Elliott, Arkansas Experiment Station.

*Secretary*, J. A. McClintock Georgia Experiment Station.

*Member of the Council of the American Phytopathological Society for 1921*, H. R. Rosen, Arkansas Experiment Station.

J. A. Elliott,
*Secretary.*

[Phytopathology for June (10: 321–352, Plate XII) was issued June 12, 1920.]

PHYTOPATHOLOGY

VOLUME X NUMBER 8

AUGUST, 1920

# A PESTALOZZIA PRODUCING A TUMOR ON THE SAPODILLA TREE

Nellie A. Brown

With Five Figures in the Text

## THE HOST

The sapodilla (*Achras zapota* Linn.) is one of the best indigenous fruits of the American tropics. The tree is cultivated in southern Florida, and grows wild on many of the Florida Keys. It is evergreen, with stiff glossy leaves, and a dense rounded crown, and reaches a height of 25 to 50 feet. The wood is hard and durable. The fruit has a variety of forms: round, conical, or globose depressed, 2 to 3½ inches in diameter. The skin is thin and rusty brown in color, the flesh translucent, dark tan color, sweet, juicy, and delicious when well ripened. It is a great favorite in Florida, and is shipped successfully to New York City. In Florida this tree seems to be remarkably free from fungous diseases.

## THE DISEASE AND ITS CAUSE

The disease reported in this paper occurred in Buena Vista, Dade County, Florida. It was found by Prof. P. H. Rolfs, Director of the Florida Experiment Station, who stated that the various tumors on the tree were from one year to eight years old. He considered the disease contagious but not enough so to infect surrounding trees, as only one other tree in the neighborhood had shown similar growths. The stem sent by him had a tumor 2 inches in diameter (fig. 1). This growth was rather smooth on the surface, which was partly covered with lichens and algae. There were also many tiny black pycnidia, but, like the lichens and algae, they were present on other parts of the stem and so had no significance. The gall appeared brown and dry but in cutting through, a soft portion was found outside the wood, which indicated that it was still alive. The gall is of the hard type composed mostly of wood covered with an irregular layer of thickened bark. The cortex and the wood were examined carefully for bacteria or fungi and a fungus was found penetrating the cell. No fruiting bodies were seen. Pieces of the gall were sterilized and placed in a moist chamber and after a few days acervuli appeared containing Pestalozzia spores. Numerous isolations were made from the tumor by agar poured plates and sterile vegetable and agar tube cultures and the fruiting bodies on both plate and tube cultures were those of a Pestalozzia.

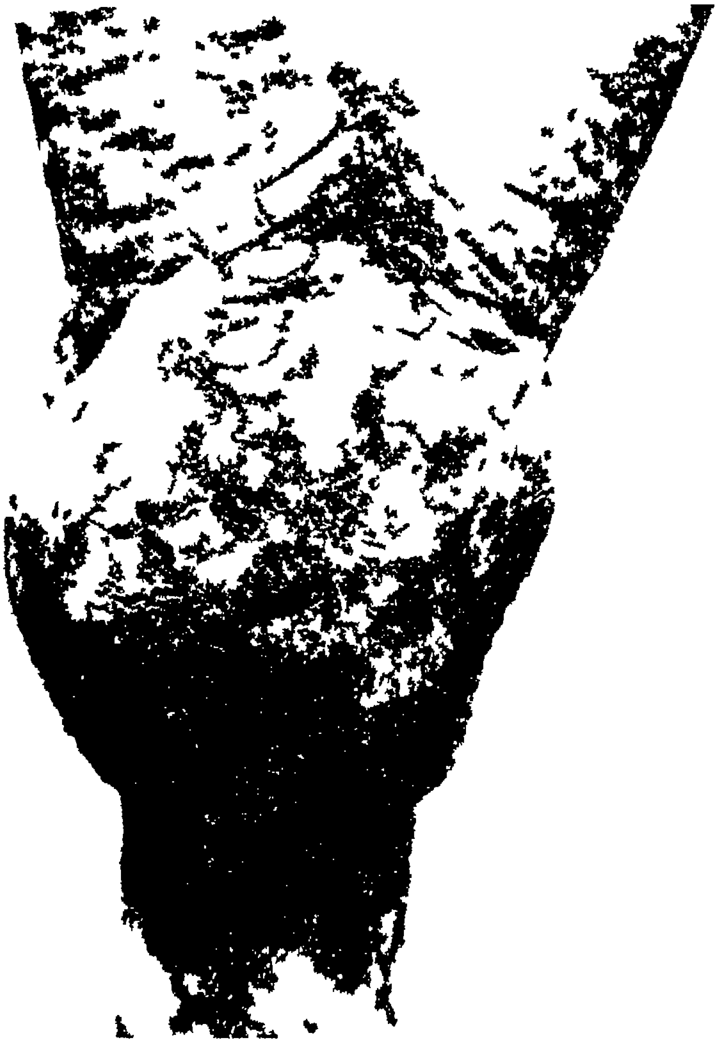

Fig. 1. Tumor Occurring on the Sapodilla Tree in an Orchard at Buena Vista, Fla.

### SPORE CHARACTERISTICS

The conidia are four-septate with three central cells darker and a little larger than the hyaline cell at either end. Of the three central cells the lower one is usually lighter than the other two and the central one darker than the one above it (fig. 2a). In young cultures, but not too young, this feature is more pronounced than in the host material or in old cultures. The apical cell has three bristles which are quite constant; occasionally there will be four. The size of the spore is 16 to 24μ by 6 to 10μ. The bristle is 16 to 26μ. When the fungus is grown for four days on sterile potato cylinders, the size of the conidia is 20 to 26μ by 5 to 6μ. The bristle is 13 to 24μ. When grown for twenty days on cornmeal agar, the size of the conidia is 16 to 30μ by 6 to 8μ. The bristle is 16 to 26μ. Dozens of mounts of spores grown on different kinds of media were studied, and cultures of different ages. In all these only a few spores were found which had five or six cross walls and an occasional one with seven or eight. The spores with six septa had six bristles, those with seven and eight had the usual number or none at all.

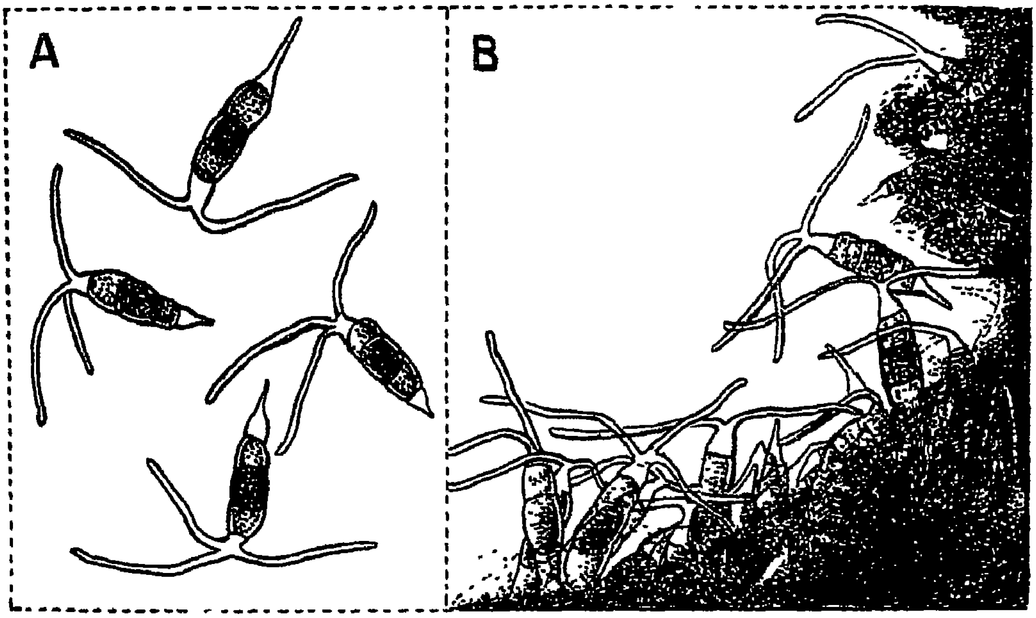

FIG. 2. *A*. Spores of Pestalozzia isolated from sapodilla tumor and grown on sterile potato cylinders. × 384.

*B*. Pestalozzia acervulus in mango tumor produced by the sapodilla Pestalozzia. × 384.

In old cultures, three months or older, spores with one dark cell and a hyaline end one with bristles sometimes occurred. No other form of fruiting body was seen.

Germination occurs usually from the lower of the three central cells, but it may occur from any other cell of the spore. Occasionally two germinating tubes are sent out from the same cell.

## INOCULATIONS

It was necessary to have some sapodilla trees to inoculate, to prove the infectiousness of the strain isolated. The southern nurseries, however, either did not have them or did not think it advisable to ship so far north, and none could be procured. Later, fruit in the market at Miami was purchased by mail and sent to Washington and the seeds from them planted. The sapodilla is a slow grower and it was necessary to wait until the seedling trees were of a fair size to inoculate. This was about a year after the isolations were made. In a month and a half small outgrowths appeared at the inoculated places on about 75 per cent of the stems. These increased very slowly in size, corresponding with the slow growth of the trees (figs. 3 and 4). The inoculations were made by smearing the spores on the stems and then puncturing with a sterile needle. The trees were kept in a greenhouse where the temperature was usually 75° to 80° F.

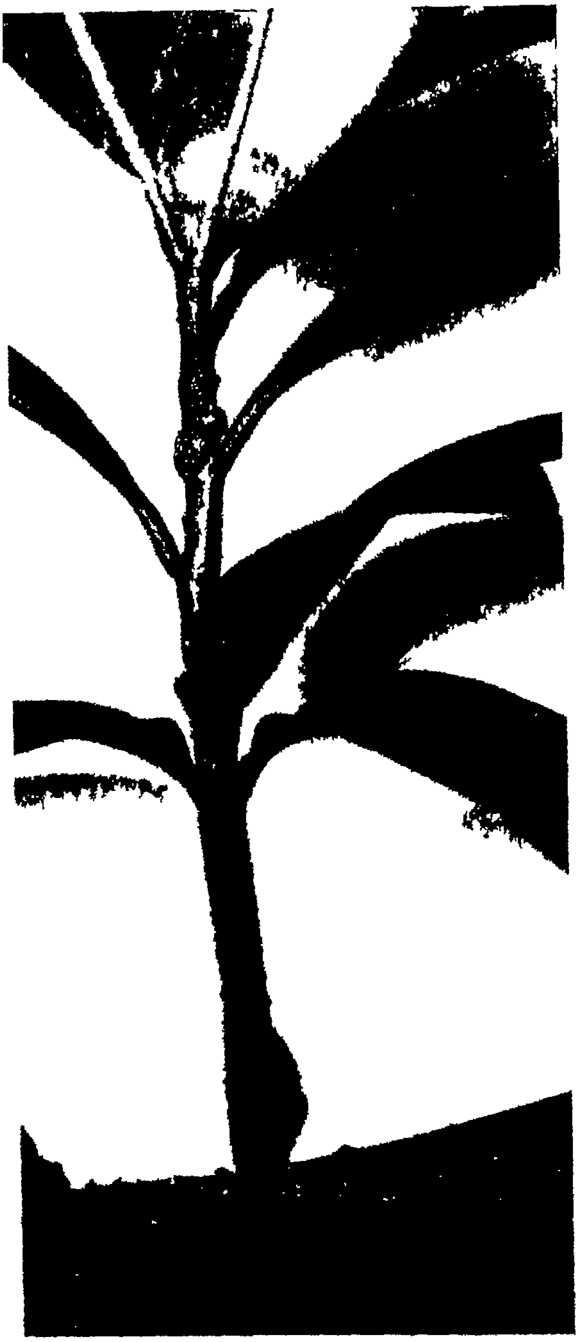

FIG. 3. A YOUNG SAPODILLA TREE WITH TUMORS PRODUCED BY INOCULATING WITH THE SAPODILLA PESTALOZZIA NOVEMBER 11, 1915
Photographed March 14, 1916

Two isolations were tested out, one which produced an abundance of spores, the other at first a meager amount of spores. This latter isolation proved to be the more infectious. One thing of interest was noted during the inoculation work; the very youngest and most tender tissue did not become infected as readily as the older tissue. The stems, however, were all comparatively young and not as yet toughened, but the stems at the growing point did not produce outgrowths. However, reddish brown spotting of very young leaves of the sapodilla occurred, while the toughened leaves remained unchanged when inoculated.

When the galls were about a centimeter in diameter the fungus was reisolated and other small sapodilla trees inoculated with the reisolation colonies. In due time galls formed on these trees also, but only about half of the inoculations proved infectious.

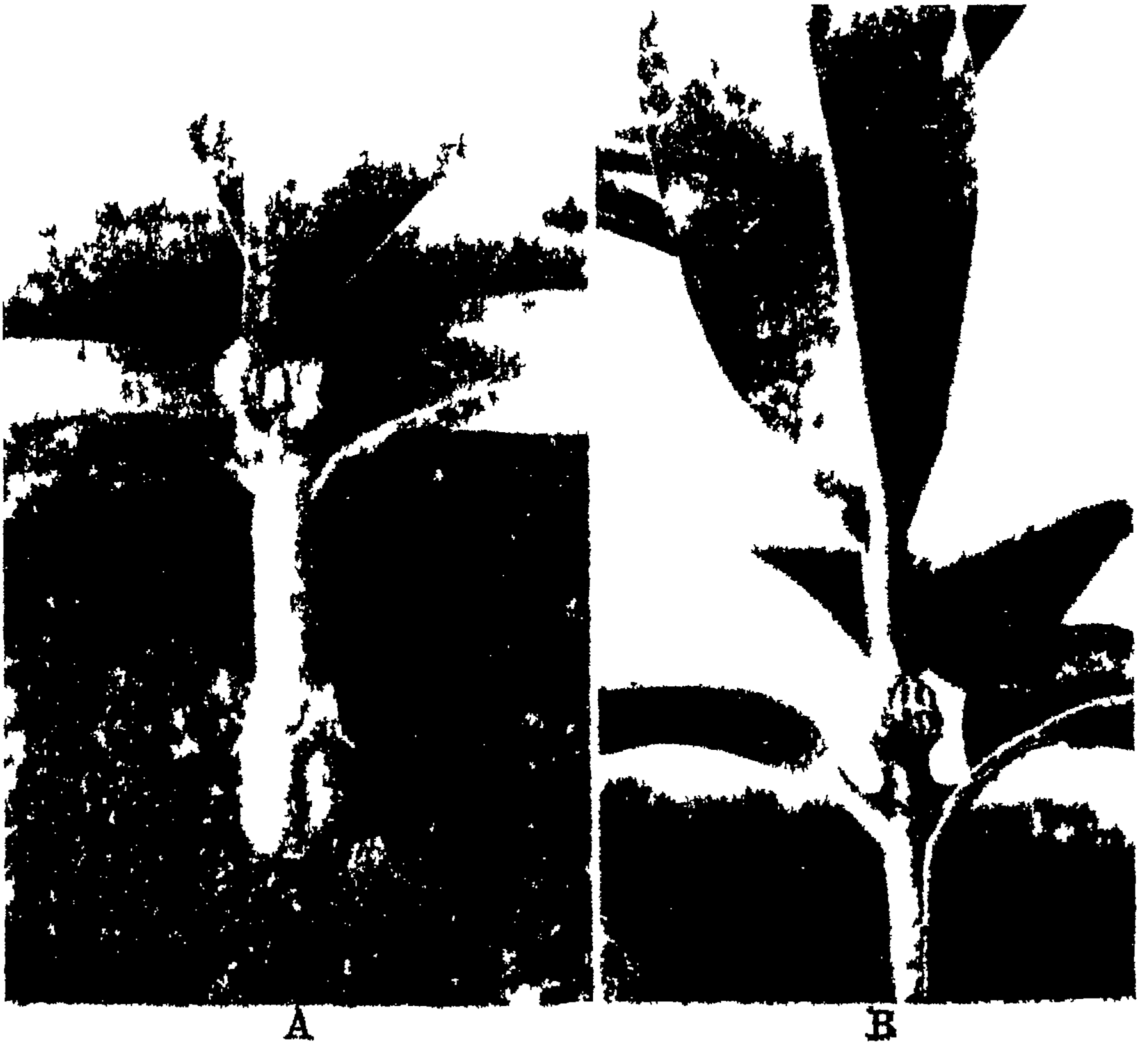

FIG. 4. A. Young Sapodilla tree with tumor produced by inoculating with the sapodilla Pestalozzia November 11, 1915. Photographed July 13, 1916.
*B.* Young sapodilla tree with tumor produced by inoculating with the sapodilla Pestalozzia November 11, 1915. Photographed March 1, 1916.

### INOCULATIONS INTO OTHER PLANTS

Inoculations were also made into other plants and trees, some of which are susceptible to the crown gall organism, *Bacterium tumefaciens*. Rose bushes, sugar beets, Paris daisy, citrus trees, pelargonium, avocado, begonia, cacao, Aleppo pine, white pine, spruce, hemlock, larch, balsam, olive and mango were inoculated, several tests being made with some of them. No outgrowths formed on any except the mango (*Mangifera indica* Linn.), olive (*Olea europaea* Linn.) and balsam (*Abies balsamea* Linn.). Small galls 3 to 10 mm. in diameter formed on the olive and balsam in the course of a year and large galls 1 inch in diameter on the mango. In mangoes grown in greenhouses swellings at nodes frequently occur. These nodal swellings need not be confused with the tumors produced by the fungus as they are easily distinguished.

FIG. 5. TUMOR ON MANGO TREE

Tree inoculated July 10, 1916, with sapodilla Pestalozzia. Photographed June 28, 1918.

The tumor on mango grew very slowly, in fact no definite outgrowth had taken place in three months. In six months there was a typical tumor encircling the stem as is the case in the sapodilla. This growth continued slowly for two years when it had reached a diameter of 2 inches (fig. 5).

One of the mango tumors was cut off three years after the tree was inoculated, part of it placed in a moist chamber and the other part used successfully for reisolating the fungus. In a few days the portion in the moist chamber showed the acervuli breaking through the surface of the gall and the typical Pestalozzia spores appearing (fig. 2, B). The fungus was also isolated from the olive gall.

### INOCULATIONS INTO CONIFERS

The fungus was also infectious to the larch (*Larix occidentalis* Nutt.), blue spruce (*Picea pungens* Englm.), and hemlock (*Tsüga canadênsis* Sarg.), producing a blight instead of outgrowths. The blighting occurred in young leaves when the spores were sprayed on them. No wounding was necessary. This condition was carried to other young leaves as they continued to appear during the summer. The check plants were not infected. Several attempts were made to infect the white pine in the same way, with negative results. The trees used were young, none of them being over 16 inches tall. They had been kept in pots out of doors during the winter, then in early spring brought into the greenhouse and repotted. At the time of inoculation, young leaves were growing freely. After inoculation, some of the trees were put in an infection cage while others were kept in the same greenhouse in the open. This was done to see if the excessive moisture of the cage would favor infection. The temperature of the greenhouse ranged from 72° to 78° F.

The trees were inoculated in two ways: in one case the spores were washed off the cultures into sterile water and the plants sprayed with an atomizer, some of them being wounded by needle pricks; in the other the spores were smeared on the stems and leaves and wounds made by puncturing with a delicate sterile needle.

No galls were produced on any of the trees, but a very decided blight occurred in the young larch leaves, a fair amount of blight in the young leaves of blue spruce, and a trace on the hemlock. The inoculations of these three were as follows:

Two larch trees 16 inches tall were inoculated, one being sprayed and wounded, the other sprayed only. Both were kept in an infection cage for five days. Blighting of the leaves occurred in both trees two weeks after they were taken from the infection cage. The check in the open remained healthy. Razor sections showed fungus mycelium penetrating the cells, and when blighted leaves were placed in a moist chamber, the Pestalozzia spores were produced in abundance. This blighting continued as other young leaves pushed out during the summer. The wounded tree was affected somewhat more than the other.

Three blue spruces were inoculated. Two were sprayed with spores without wounding, then placed in an infection cage; the other tree was smeared with spores and punctured, but not placed in a cage. Of the two sprayed, only one became infected. The one punctured and not kept in the cage became infected slightly more than the other. Evidently the conditions of moisture both in and out of the infection cage were sufficient to infect, and the wounding helped to increase infection.

Only one hemlock tree 12 inches tall was available; it was sprayed but not punctured, then kept in the infection cage for five days. In two weeks there was slight blighting but this did not continue to advance.

The inoculations into the conifers were made because of the infectious character of *Pestalozzia funerea* to white pine (*Pinus strobus* L.), Norway spruce (*Picea excelsa* Link), and hemlock (*Tsúga canadénsis* L.), as demonstrated in the work of John J. Wenner (13), in which he shows that seedlings one month old to three-year-old trees under certain conditions of moisture are blighted by this species, and because *Pestalozzia hartigii* has been recognized as the cause of a seedling disease of the spruce and the silver fir in Europe (1), (3), (11), (12).

The balsams (*Abies balsámea* L.) were inoculated more especially because of a tumor reported by P. Hennings (5) on *Abies balsámea*, produced by a Pestalozzia which he has named *Pestalozzia tumefaciens*. The galls resulting from needle-prick inoculations into balsam with the sapodilla isolation were very slow-growing and small, the largest one reaching hardly more than a centimeter in diameter in a year and a half. The size is about the same or a little larger than those in the Pathological Collection of the United States Department of Agriculture, produced by *Pestalozzia tumefaciens* (P. Hennings). However, in his description of this particular out-growth which appears in different species of Abies, Hennings states that they reach a size of from 1 to 5 centimeters in diameter, and a badly attacked tree must be cut out and burned in order to check the spread of the disease.

## COMPARISON OF THE SAPODILLA PESTALOZZIA WITH OTHER TUMOR-FORMING PESTALOZZIAS

The spores of *Pestalozzia tumefaciens* do not agree in size and number of cells with the Pestalozzia isolated from the sapodilla. There are other differences, e.g., in color of central cells and shape of end cells. *Pestalozzia tumefaciens* is 13 to 17$\mu$ long by 5 to 6$\mu$ wide, bristle 25$\mu$. Spores are cylindrical-oblong with three cross walls, making 4 cells. The two middle cells are large and dark brown in color, while those at each end are colorless and somewhat papillary. The upper cell bears three very fine, spreading, colorless bristles.

Nor do the spores or the sapodilla Pestalozzia agree with those of *Pestalozzia gongrogina* (F. Temme) (10), which according to Temme produces a gall on *Salix viminalis*. These willow galls grow from year to year, the largest ones becoming the size of a hen's egg. They are soft and green at first and later become woody; in the course of a few years the trees are covered with various sizes of galls. This description is not unlike that of the galls on willow due to the crown-gall organism, *Bacterium tumefaciens*. P. Hennings became interested in the tumors on *Salix viminalis*, and found that the disease was infectious to other varieties of willow. Neither Hennings nor Temme produced galls by inoculations with pure cultures of *Pestalozzia tumefaciens* or *Pestalozzia gongrogina*. In his description of the latter, F. Temme states that spores are produced in a definite pycnidium. They are slightly bent, club-shaped, 0.024 mm. long by 0.004 mm. wide, 3-celled, colorless, with a delicate curved bristle at upper end.

Mr. I. B. Pole Evans and Miss Ethel M. Doidge (2) of the Transvaal Department of Agriculture found a Pestalozzia on *Cydonia vulgaris*. Warty outgrowths formed by larger numbers of arrested adventitious roots, they claimed, were produced by this fungus. As the spore has but one bristle it is unlike that of the Pestalozzia considered in this paper. Evans and Doidge have named their fungus *Pestalozzia* (Monochaetia Sacc.) *cydoniae*.

### ANOTHER PESTALOZZIA ON A RELATED HOST

There is a *Pestalozzia sapotae* (P. Henn.) n. sp. reported from Brazil on the leaves of *Achrae sapotae* (4). The spots are dark, wide spreading, with acervuli on the upper side of leaf bursting out black. Conidia spindle shaped, 4-septate; three cells black, 10 to 14μ by 5 to 6μ, end cells hyaline; papillate, three filiform bristles 10 to 15μ, pedicel short and hyaline.

As there has been confusion regarding the botanical name of the sapodilla, the *Achrae sapotae* mentioned above may or may not be identical with *Achras zapota*, the tree on which the tumor occurred. The description of the Pestalozzia producing the spotting of the leaves does not correspond to that of the Pestalozzia producing the tumors on *Achras zapota*.

Leaves of sapodilla inoculated with the sapodilla tumor Pestalozzia produce rather reddish, well defined spots, and the conidia in these spots maintain the typical characteristics of those produced in the tumor.

As there are essential differences between the spores of the Pestalozzia forming the tumor on the sapodilla and those forming tumors on the other plants, namely, balsam and willow, the tumor-forming Pestalozzia of sapodilla cannot be classed with either of them. It resembles *Pestalozzia funerea* in some respects (8), i.e., the spores are very much like the funerea spores, it is pathogenic, and it also produces a blight on some of the members of the pine family. However, *Pestalozzia funerea* is not known to produce tumors. Comparison was made with spores of *Pestalozzia guepini* Desm., a species known to be parasitic, but there were well defined differences between the two (9). The descriptions of other species reported to be parasitic were studied, but no agreement was found between them and that of the sapodilla species (6) (7). It seems best, therefore, to consider this fungus as a separate species, and because of its tendency to produce hard galls, the name *Pestalozzia scirrofaciens* is suggested and the following characterization given:

### Pestalozzia scirrofaciens n. sp.

The acervuli are immersed, breaking out to the surface and of a black color after the gall has been kept in a moist chamber a few days. The conidia are ovate-oblong to spindle shape, slightly curved, 4-septate with slight or no constriction of the septa. The three central cells are olive brown, the terminal cells colorless, conoid, bearing at the apex three, rarely four, slender bristles bent outward, the pedicle is short and slender. Size of spores 16 to 24$\mu$ by 6 to 10$\mu$. Bristle 16 to 26$\mu$.

Habitat. In stems of *Achras zapota*, Florida, producing a hard tumor.

## FAVORABLE MEDIA FOR GROWTH OF THE FUNGUS

Sterile potato cylinders are a very good medium for the growth of this species, and the fungus will continue to live thereon over a year and a half but does not live two years, temperatures being 12° to 17°C. Carrot and potato agar are also good. There is very little mycelium produced on cornmeal agar, but it is luxuriant on potato cylinders and sugar-beet agar. Acervuli are produced in abundance on plain potato cylinders, and occasionally black spore masses protrude in a horn like shape. Weak acids such as 1 and 2 per cent malic acid added to potato cylinders retard the growth of the mycelium and lessen the production of spores.

In three to seven days after transferring to potato cylinders, small, black dots appear on the surface, and later black, liquid-like drops exude from them. One of these drops examined under the microscope was found to be made up of masses of conidia. When young, these small, black bodies have a structure which to all appearances is a pycnidium. As development goes on, however, they break open across the top and we have acervuli extruding spores.

## CONTROL

The control method which proved successful in the Buena Vista orchard where the disease was found is the one to be recommended; namely, cutting down and burning the infected trees. This method was used successfully with trees infected by other gall-producing Pestalozzias.

## SUMMARY

Tumors of various sizes occurred on stems of the sapodilla tree growing in an orchard in Dade County, Florida.

A Pestalozzia was isolated from one of these outgrowths, and inoculations into young sapodilla trees produced slow growing tumors from which the fungus was reisolated.

Small galls were produced on the balsam fir and olive, and large ones on the mango tree.

Inoculations into larch, hemlock, and blue spruce produced blighting of the leaves but no tumors. No blighting occurred on the white pine.

The Pestalozzia isolated from the sapodilla tumors does not agree with *Pestalozzia tumefaciens,* which produces tumors on different species of Abies. Neither does it agree with the Pestalozzia reported to produce tumors on different species of Salix.

It agrees in some respects with *Pestalozzia funerea* Desm., which is a parasitic species but one not known to produce galls.

The name *Pestalozzia scirrofaciens* is suggested.

The disease can be controlled in an orchard by destroying the infected trees.

BUREAU OF PLANT INDUSTRY
WASHINGTON, D. C.

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# BRIEFER ARTICLES

## NOTES ON PERIDERMIUM HARKNESSII

George G. Hedgcock and N. Rex Hunt

*Peridermium harknessii* Am. Auct. as used in this paper applies to the gall-producing form of Peridermium which is the aecial stage of *Cronartium coleosporoides* (Dict. & Holw.) Arthur. This species attacks both *Pinus contorta* and *Pinus ponderosa* in some regions in the western and northwestern states, more especially the former species of pine in Colorado and Wyoming. The galls produced by this species vary in shape from fusiform swellings on recently infected small twigs to sub-spherical galls from older infections on limbs, twigs, and small trunks which are encircled by the diseased tissues. Infections on one side of a trunk or limb may produce a hemispherical form. Young trees often are either killed or stunted by the strangulating effect of these galls.

Rodents, especially squirrels and porcupines, feed upon the cambial layer of the galls of *Peridermium harknessii*, owing to the sweet taste of this layer when either aecia or pycnia are present or in process of formation. The senior writer has repeatedly observed them feeding near Fox Park, Wyoming, on galls of *Pinus contorta*. On a gall defaced by these rodents, the wood dries out and the bark is not replaced. Frequently new gall tissue forms in a fold adjacent to the wound, producing an appearance commonly called "cat-face" in this region. In a timbered area being cut over near Fox Park, it was noted that both *Trametes pini* (Brot.) Fr. and *Polyporus schweinitzii* Fr. frequently gain entrance through such wounds.

The action of these rodents in feeding not only on the galls of this Peridermium, but also on the healthy wood of adjacent small trees, suggested to the writers the possibility of aecial infection taking place through their depredations. This was strengthened by observations on the spread of the Peridermium in some localities in this region in the apparent absence of telial hosts beneath the trees.

On April 20, 1912, six trees of *Pinus sabiniana* were inoculated by inserting in a wound under the bark a small piece from the cambium of the immature fruiting portion of a live gall of *Peridermium harknessii* on *Pinus attenuata* collected by Dr. E. P. Meinecke in California, April 12, 1912. The wounds were tightly wrapped with wet cotton. Six control trees were similarly wounded and wrapped. Two out of the six trees inoculated developed typical galls of *Peridermium harknessii* from these graft inoculations but died before aecia were produced.

On June 26, 1915, forty-four trees of eleven species of pine were inoculated as follows: Fruiting galls of *Peridermium harknessii* from *Pinus contorta*, collected by Ellsworth Bethel at Tolland, Colorado, June 20, were held over a sheet of paper and the aeciospores dislodged by jarring the galls. Wounds were made by inserting a scalpel under the cambium of the pine trees to be inoculated, then spores were introduced in the wounds, which were then tightly wrapped with strips of wet cotton.

Of the trees inoculated, sixteen, or 36 per cent, became infected at the point of inoculation. Oblong galls were formed. Of these infected trees, more than half have since died from the effects of the disease. Four trees, all of *Pinus ponderosa*, produced aecia on the galls, three in 1917, and one in 1918. No pycnia were observed.

At the time of inoculation aeciospores taken from those used in the experiment were germinated on the surface of sugar agar poured plates. Of the spores that germinated, all produced the usual aecial mycelium, and none the promycelium characteristic of teliospores.

The number of trees inoculated of each species, and the number infected was as follows: *Pinus caribaea*, six inoculated and three infected; *P. contorta*, six and four; *P. halepensis*, one and one; *P. mayriana*, six and two; *P. palustris*, two and none; *P. pinea*, one and one; *P. ponderosa*, two and none; *P. ponderosa scopulorum*, nine and four; *P. sabiniana*, two and one; *P. taeda*, six and none; and *P. virginiana*, three and none.

Two other similar experiments have been performed, one with aeciospores of *Peridermium harknessii* from *Pinus ponderosa*. A few galls were obtained, mostly on *Pinus ponderosa*, but none fruited. One tree of *Pinus virginiana* was infected, which is an additional species to those named above.

These experiments were all performed with potted plants grown from the seed under controlled conditions in the green-houses at Washington, D. C., and apart from other experiments with gall-forming species of Peridermium. Control trees of each species of pine used remained free from infection. The telial stage of *Cronartium coleosporoides* has never been found in Colorado and Wyoming, antedating or coincident with the aecial. This precludes a possibility of the presence of sporidia on the galls.

*Peridermium harknessii* has been collected in nature on the following species of pine in the United States, the data being taken from collections in this office at Washington, D. C.: On *Pinus attenuata* Lemm., two collections from California; on *P. contorta* Loud., two from California, fifty-one from Colorado, eight from Idaho, eight from Montana. six from Oregon, one from South Dakota, one from Washington, and twelve from Wyoming; on *P. jeffreyi* "Oreg. Comm.," two from California; on *P. ponderosa* Laws., three from California, four from Idaho, and two from Montana; on *P. ponderosa scopulorum* Engelm., six from Colorado, one from Nebraska, and three from South Dakota.

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# PYTHIACYSTIS AND PHYTOPHTHORA[1]

H. S. FAWCETT

The following brief discussion is given here in order to correct, as far as possible, any confusion in regard to the distribution of *Pythiacystis citrophthora* that the author's previous publications may have left. The writer has previously reported finding on Citrus, in Florida, Cuba and the Isle of Pines, a fungus that he then considered to be *Pythiacystis citrophthora* (1). The cultures on which this report was based, though differing somewhat from those of *Pythiacystis citrophthora* Sm. and Sm. found in connection with lemon brown rot (2) and in connection with one type of gummosis (3), were thought to be sufficiently similar to be considered a variety or strain of the above mentioned species. The sporangia and zoospores and also the mycelial growth at certain temperatures were similar in appearance to *P. citrophthora*. There appeared also a close similarity in their behavior as compared to *P. citrophthora*, when inoculated into citrus bark. Later it was found that the growth temperature relations were markedly different from that of *P. citrophthora* (4). The behavior of the Florida-Cuba cultures in the rotting of fruit was also seen to be slightly different from that of *Pythiacystis citrophthora* in California, and yet the similarity in the appearance of the rot produced was more striking than were the differences.

In order to correct any misunderstanding that may have arisen as to the identity of the fungus reported upon in the first mentioned publication, the writer wishes to state that the cultures which he found in connection with mal di gomma in Florida, Cuba and the Isle of Pines were all apparently *Phytophthora terrestria* Sherbakoff (5), and not typical *Phythiacystis citrophthora*. Sherbakoff has kindly examined those from Florida and Cuba, and considers them to be identical with his fungus. Stevens also (6) has made an extensive cultural examination of diseased bark in connection with mal di gomma in various parts of Florida, and has repeatedly isolated a fungus which appears to be identical with *P. terrestria.* It may also be of interest here to state that a culture which appears to be identical with *P. terrestria* was isolated by the writer from diseased bark at the base of a young orange tree from Lindsay, California. This, so far as is known, is the only time this form has been obtained from California specimens. Most of the California strains obtained from fruits and diseased bark of citrus do not readily show the perfect stage in cultures, and are like *Phythiacystis citrophthora* as described by Smith and his co-workers. Cultures obtained from Arizona specimens of young orange trees killed by gummosis were of the *Phythiacystis citrophthora* type. A Phythiacystis-like fungus from citrus received by the writer from George Fawcett from Tucuman, Argentine Republic, when submitted to Sherbakoff was thought by him to be nearly if not entirely identical with *Phytophthora terrestria.*

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[1] Paper No. 69, University of California, Graduate School of Tropical Agriculture and Citrus Experiment Station, Riverside, California.

A number of different Phythiacystis-like forms have been found in California on other hosts. Barrett (7) has reported on some of these and has suggested that some of them, at least, are probably true Phytophthora. *Pythiacystis citrophthora* itself has so many cultural characteristics in common with known Phytophthora, such as *P. terrestria*, for example, that one is led to suspect that if its perfect stage were known it would prove to be closely related to the above species.

The author (8) also reported a Pythiacystis-like fungus affecting avocado trees. A further study of this culture indicates that it is probably a Phytophthora, and quite different in some of its cultural characteristics from either *Pythiacystis citrophthora* as here considered, or *Phytophthora terrestria.*

To summarize, most of the cultures so far made from fruit and from the diseased bark of one type of gummosis of citrus in California appear to be *Pythiacystis citrophthora* proper as described by Smith & Smith. In one instance only has *Phytophthora terrestria* been found on citrus in California. Cultures made from diseased bark of mal di gomma (footrot) specimens of citrus in Florida, Cuba, the Isle of Pines and the Argentine Republic all appear to have been *Phytophthora terrestria* as described by Sherbakoff. Other Pythiacystis-like forms so far obtained from hosts other than citrus in California appear to be different from either of the above species.

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# SOME METHODS FOR INVESTIGATING INTERNAL SEED INFECTION

J. B. S. NORTON AND C. C. CHEN

The technique for the study of diseases carried by internal seed infection has never been standardized, for the methods are still in the experimental stage and no extensive or systematic investigation of seed diseases has yet been made. However, a good general technique will be needed when such studies are made. In the course of investigations on internal seed diseases, the writers have developed a technique that gives rather successful and practical results. This has been developed from the method used by Selby and Manns[1] in their studies on diseases of cereals and grasses. Our method is outlined as follows:

1. Soak the seeds to be studied in water from ten to twelve hours. The presoaking is to render the spores of pathogens on the seed coats more susceptible to the action of the disinfectant, and at the same time to prevent seed injury by the disinfectant. The efficiency of this treatment has been tested out also by Braun.[2]

2. Shake the presoaked seeds from three to five minutes in an alcoholic solution of corrosive sublimate (2 grams to 1000 cc. of 50 per cent alcohol) in a sterilized test tube.

3. Remove the traces of the disinfectant from the seeds by first washing once in ethyl alcohol (93 per cent) and then three times in sterile water. The writers have determined by a series of experiments that the latter operation is absolutely necessary in order to prevent the alcohol

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[1] Selby, A. D., and Manns, T. F. Studies in diseases of cereals and grasses. Ohio Agr. Expt. Sta. Bul. 203: 213–218. 1909.

[2] Braun, H. Presoaking as a means of preventing seed injury due to disinfectants and of increasing germicidal efficiency. Science, n.s., 49: 544–545. 1919.

from injuring the seeds. In the tests summarized in the following table, one hundred seeds of each kind, presoaked in water for ten hours, were used for each trial. They were given the standard germination test in the State Seed Laboratory. The table shows the details and results.

| TREATMENT | PERCENTAGE OF GERMINATION | | | |
|---|---|---|---|---|
| | Wheat | Corn | Peas | Soy-beans |
| No treatment .. ........ ....... ....... ... .. .. | 72 5 | 88 0 | 92 | 92 5 |
| Immersed in corrosive sublimate (alcoholic) solution for five minutes . ........... ......... ..... .. | 0 | 74 5 | 58 | 89 0 |
| Immersed in corrosive sublimate (alcoholic) solution for five minutes and washed in 93 per cent alcohol . . | 0 | 20 0 | 11 | 53 0 |
| Immersed in corrosive sublimate (alcoholic) solution for five minutes; then washed in 93 per cent alcohol; then washed in water three times.................. ...... | 69 5 | 91.0 | 77 | 90.5 |

The percentages in the table represent the average of two duplicate tests for each kind of seed.

Probably the alcohol washing could be eliminated, but it is desirable to remove any chance of interference with the growth of internal organisms.

4. Pick out the surface-sterilized seeds with sterile forceps and plant them in sterile nutrient agar plates for germination, during which many of the pathogens carried internally will develop sufficiently to be recognized and can then be isolated for further examination.

Or, plant the surface sterilized seeds in large, thick test tubes containing absorbent cotton saturated with a sterile nutrient solution. This practice enables the investigator to discover the presence of parasites carried internally by the seed, and to actually determine their effect upon the young plant. The nutrient solution enables the seedlings to continue their growth, so that death from lack of nourishment may not be confused with the action of parasites. The cultures may be placed in a moist chamber to aid germination.

5. Make pure cultures from the organisms developing from the infected seeds, and inoculate healthy plants with the isolated organisms both in tube cultures and in pot cultures in sand.

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## REVIEWS

A noteworthy contribution to the literature on tropical plant diseases is "The Red Ring or 'Root' Disease of Coconut Palms," by William Nowell, published in the W. I. Bul., vol. xvii, no. 4, 1919 (see also Agric. News, p. 398, December 14, 1918, and Agric. News, p. 46, February 8, 1919).

Up to the time of Nowell's work on this disease it had been reported many times but usually referred to as root disease, merely intending to indicate that, supposedly, the trouble arose in the roots or in the soil. The history of the disease is given, and its distribution as found by Nowell in Trinidad and Tobago. To this might be added Panama and Cuba as observed by the reviewer.

The external symptoms are a progressive yellowing and browning of the leaves from below upwards, commencing at the tip of each leaf. The internal symptoms consist of a red zone in the stem, which is some distance from the bark and, at least at the base, forms a complete circle.

The red tissues are reported always to be heavily infested with nematodes which Cobb has described as a new species, *Aphelenchus cocophilus.* This fact and the fact that much careful work has failed to demonstrate the presence of either bacteria or fungi have led to the conclusion that the nematode is the cause of the trouble.

This disease has been and is serious in certain places, destroying great numbers of trees, usually about the age when they come into bearing.

No specific remedy is proposed, but experiments are being conducted by Nowell looking to the control of this pest. At least a great advance has been made in demonstrating the presence of a pest in the diseased tissues.

Nowell's theory as to a possible manner of infestation is of interest and warrants further investigation. The theory is that the nematodes may occur in the seed bed, there infest the young plant about the base of the leaves, and from that point work downward as the trunk develops. This is borne out by the fact that occasionally red ring is found in the trunk when the roots are free from the pest.

JOHN R. JOHNSTON.

# PHYTOPATHOLOGICAL NOTES

*Personals.* Dr. Edgar W. Olive has resigned as Curator in the Brooklyn Botanic Garden. He has gone into private business, his address being 904 Hume-Mansur Building, Indianapolis, Ind.

Dr. George L. Peltier has given up his position at the Alabama Agricultural Experiment Station and accepted an appointment as pathologist at the Nebraska Agricultural Experiment Station.

Dr G. R. Bisby, assistant pathologist at the University of Minnesota, has been elected pathologist in the Manitoba Agricultural College at Winnipeg. Dr. Bisby assumed his new duties June 1.

Mr. A. F. Thiel, who has been stationed at St. Paul, Minnesota, on rust epidemiology studies, was elected assistant pathologist at the Alabama Polytechnic Institute, Auburn, Alabama. Mr. Thiel began his duties at Alabama on April 1.

Dr. Julian G. Leach, associate professor of botany in the Colorado Agricultural College, has been appointed assistant pathologist at the University of Minnesota.

Professor Frank T. McFarland, University of Kentucky, has been granted a year's leave of absence. He has taken up graduate work in the Department of Plant Pathology of the University of Wisconsin.

# LITERATURE ON PLANT DISEASES[1]

COMPILED BY ALICE C. ATWOOD AND FLORENCE P. SMITH, BIBLIOGRAPHICAL ASSISTANTS, BUREAU OF PLANT INDUSTRY

## May, 1920

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All authors are urged to cooperate in making the list complete by sending their separates and by making corrections and additions, and especially by calling attention to meritorious articles outside of regular journals. Reprints or correspondence should be addressed to Miss E. R. Oberly, Librarian, Bureau of Plant Industry, U. S. Dept. Agric., Washington, D. C.

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PHYTOPATHOLOGY

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# RELATION OF CLIMATE TO THE DEVELOPMENT AND CONTROL OF LEAF ROLL OF POTATO

PAUL A. MURPHY AND E. J. WORTLEY

WITH ONE FIGURE IN THE TEXT

Prior to the pioneer work of Quanjer and Oortwijn Botjes (2) in Europe and that of the authors (1) in America, in which the infectious nature of leaf roll was proved, there was a tendency to over-emphasize the influence of environment on the disease. It was held that the presence of leaf roll in hot, dry regions was in some way attributable to the climate, leaving it a difficult question to account for the occurrence of the disease in parts of Canada in which the seasons are comparatively cool and moist. The prevalence of neighbor-infection at Charlottetown, Prince Edward Island, (Murphy and Wortley, loc. cit.) furnished a satisfactory solution of the latter difficulty, it being shown that once leaf roll seed is introduced the spreading-power of the disease is such as to account for its persistence and even for its increase from year to year under the favorable conditions of Prince Edward Island. There remained to be explained why it is that leaf roll is a factor of so much greater economic importance in warmer districts than in the cool climate of the Maritime Provinces.

## RESULT OF PLANTING SISTER TUBERS IN PRINCE EDWARD ISLAND, BERMUDA AND SOUTHERN ONTARIO

An experiment was begun in 1917 to test the effect of the climate of Prince Edward Island, Bermuda and Southern Ontario on the development of leaf roll. A crop of Garnet Chili potatoes which had a considerable percentage of leaf roll was selected in Nova Scotia in 1916, and 238 individual plants were dug which yielded three or more tubers. The tubers from each hill were given the same number and one of each was sent to Charlottetown, Bermuda and Ottawa, where they were planted in tuber units. The results are shown in table 1. As there was considerable loss from rot and other causes, about half the hills were not represented at Ottawa, but there was practically a full muster in Charlottetown and Bermuda.

This result is rather striking, emphasizing as it does the importance of the seed as a carrier of leaf roll. Comparing Bermuda, which is very liable to the disease, and Prince Edward Island, which is naturally free from it, it is found that only 14 plants out of 222 failed to react similarly at both places. Five of these were leaf roll at Charlottetown and healthy in Bermuda, while 9 were healthy at Charlottetown and leaf roll in Bermuda. It is not believed that there was any difference in diagnosis, nor is it likely that the plants became infected locally and showed the disease the same year. It is probable that the difference existed in the parent hill because it is not infrequent to find healthy and affected sister tubers in a diseased crop. Thus it appears from this experiment that where leaf roll appears it is to be traced almost altogether if not entirely so to infected seed and not directly to climatic conditions.

TABLE 1

*Result of planting sister tubers from a partly diseased crop in Prince Edward Island, Bermuda and Southern Ontario*

| | NUMBER OF PLANTS | CASES IN WHICH PLANTS REACTED SIMILARLY AT ALL PLACES | |
|---|---|---|---|
| | | Number | Per cent |
| Plants represented in three places.. ..... | 102 | 93 | 91 2 |
| Plants represented in two places.... . | 222 | 208 | 93.7 |

It is interesting to follow these two lots of potatoes for another year. The Bermuda crop after being harvested about May, 1917, was placed in cold storage, it being then too late for the Canadian season of that year, but it was sent to Charlottetown and planted there in 1918 near the corresponding Charlottetown seed. The latter was re-planted in a new plot hill by hill in the same order in which it came before. It was then found that 60.7 per cent of the Charlottetown seed was affected with leaf roll while 97.5 per cent of the Bermuda seed was diseased. The percentage of leaf roll in the Charlottetown lot the previous year had been 19 and the Bermuda lot was practically the same, there being only four more leaf roll plants found there than in Prince Edward Island. The subsequent difference must have arisen from infection being more successful under Bermuda conditions, and it is here that the influence of climate is seen, as a secondary factor which predisposes to the disease.

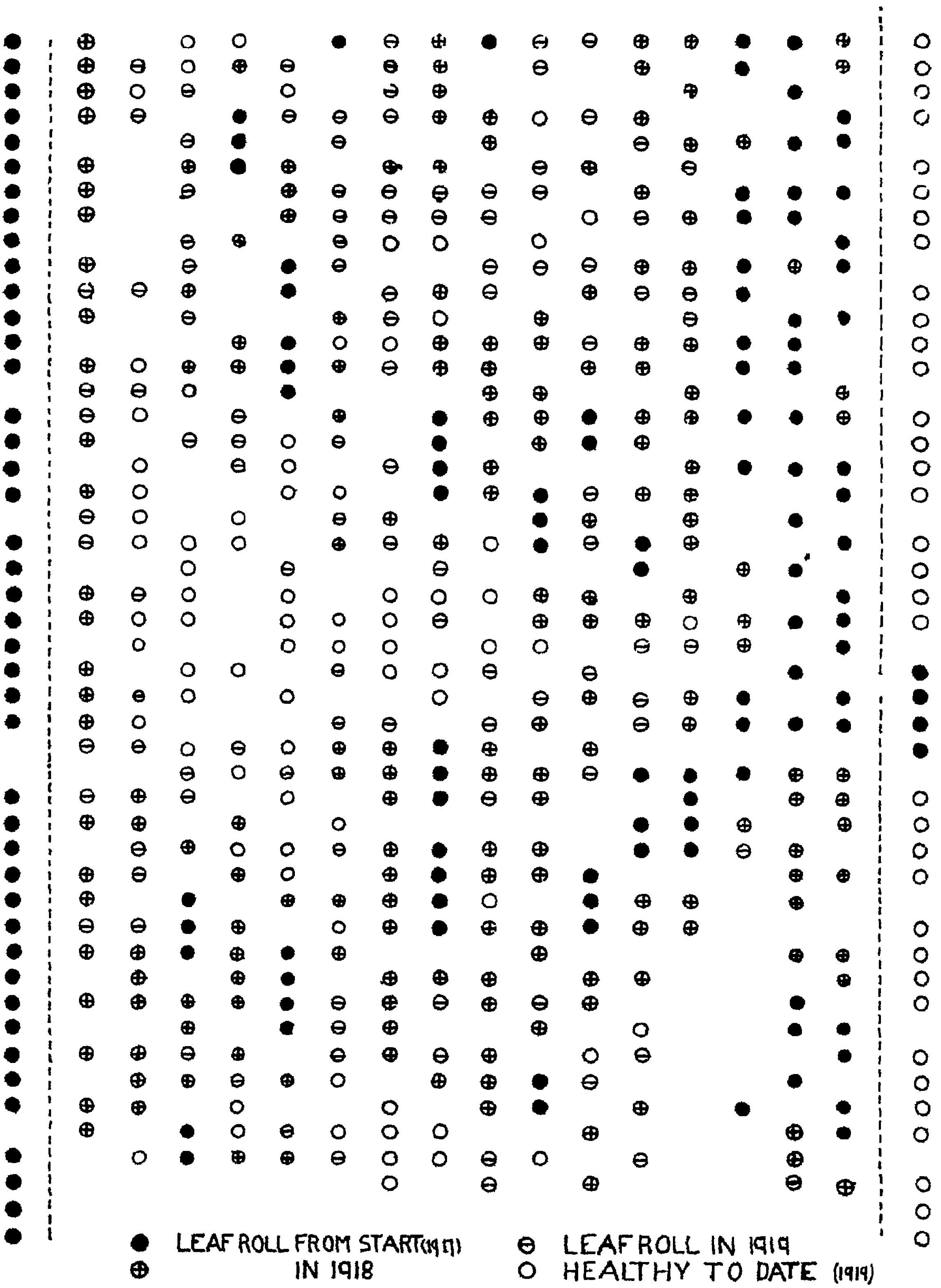

Fig. 1. The Charlottetown Plot (as it Stood for Three Years) of the Experiment in Which Sister Tubers Were Planted in Prince Edward Island, Southern Ontario and Bermuda

The circles inside the dotted lines represent plants, each of which was put back in its own place (but in a new plot) in 1917, 1918, and 1919. The plants outside the lines were present in 1917 only, as they belonged to another experiment. They are shown because they infected the outside rows particularly to the right. The spread of leaf roll from the original infection centers can be followed. The percentages of leaf roll were: 1917, 19 per cent; 1918, 60.7 per cent; 1919, 85.6 per cent.

### NEIGHBOR-INFECTION IN VARIOUS PARTS OF CANADA

These conclusions are borne out by a later experiment of the first-named author in which it was found that the spread of the disease varied greatly in different parts of Canada. Uniform seed of the variety Empire State, both healthy and diseased, was planted in the nine localities listed in table 2. The plots were so arranged that there were two rows of leaf roll plants with one row of healthy plants between them, and five rows of healthy plants outside them on both sides, thus:

H H H H H LR H LR H H H H H

Nothing in particular happened during the first year, all the plants from the healthy and leaf roll seed being healthy and leaf roll respectively.

TABLE 2

*Spread of leaf roll from diseased to healthy plants in various parts of Canada (uniform Empire State seed throughout)*

| LOCALITY | Between two rows of leaf roll | Next row to leaf roll | Second row from leaf roll | Third row from leaf roll | Fourth row from leaf roll | Fifth row from leaf roll |
|---|---|---|---|---|---|---|
| Charlottetown, P. E. I. | 15.0 | 21.2 | 2.5 | 2.5 | 0 | 0 |
| Kentville, N. S. | 35.0 | 12.5 | 6.5 | 1.2 | 2.5 | 1.2 |
| Nappan, N. S. | 20.0 | 16.2 | 0 | 0 | 0 | 0 |
| Fredericton, N. B. | 5.0 | 0 | 3.7 | 1.2 | 1.2 | 0 |
| Lennoxville, P. Q. | 17.5 | 17.5 | 0 | 0 | 0 | 3.7 |
| Ottawa, Ont. | 72.5 | 50.0 | 23.7 | 22.5 | 13.7 | 10.0 |
| Thunder Bay, Ont. | 0 | 0 | 0 | 0 | 0 | 0 |
| Brandon, Man. | 13.8 | 5.6 | 12.3 | 3.2 | 7.2 | 8.0 |
| Indian Head, Sask. | 5.3 | 1.4 | 1.4 | 0 | 0 | 0 |

Seed was saved separately from each row and planted in an experiment of the same plan in 1919. A sample from each row from most of the experiments was also grown at Charlottetown. Leaf roll appeared in the second year in varying amounts in the healthy rows (table 2, where each pair of corresponding rows is averaged), there being in general most in the center row and progressively smaller amounts in the outer ones.

It is probable that practically no dispersal took place at Brandon, Man., Indian Head, Sask., or Fredericton, N. B., in spite of the figures recorded. The inspection at these places was made under difficulties, and the corresponding samples showed no disease except 0.5 per cent at Fredericton. As to the other experiments, no infection took place at Thunder Bay in Northern Ontario, a considerable amount occurred at Charlottetown, P. E. I., Lennoxville, P. Q., and Nappan, N. S., still more at Kentville, N. S., and at Ottawa, Ont., more than twice as much as at any other place.

No entirely adequate reason can be given for these variations, although it may be significant that leaf roll is found more or less commonly in the districts around Ottawa and Kentville. There are indications that the dispersal of leaf roll varies not only from place to place but from season to season in the same locality. While at Charlottetown it has occurred regularly for three seasons, the percentage of infection contracted by healthy Garnet Chili potatoes grown between two rows of leaf roll plants being 89.5, 62.4, and 50 per cent respectively, a preliminary attempt to bring about infection in the same variety at Kentville, Nova Scotia, in 1917–1918 resulted in comparative failure, only 3 per cent of the plants becoming diseased. Nevertheless the extent to which plants are in danger from neighboring diseased ones, if only in certain years at some places, is sufficient to account for the persistence of the disease and for failure to eradicate it by the commonly recommended methods of hill selection.

## NEIGHBOR-INFECTION AND CONTROL BY MEANS OF SELECTION

When work was first begun jointly in 1915 in the affected area of Nova Scotia it was believed that leaf roll should in time eliminate itself since diseased plants produce only about one-third of a crop. The then general view was also held that the selection of the largest and healthiest hills from a partially diseased crop would hasten this process materially. Experiment and observation proved both of these views erroneous before the underlying reason, the infectious nature of the disease, was discovered.

A scheme of seed selection on a large scale was begun in coöperation with the Garnet Chili growers in King's County, Nova Scotia, where leaf roll was very common and was jeopardizing the seed trade with Bermuda. Things had come to such a pass that the Bermuda government had arranged for the transfer of some of the best stock to Maine with the purpose of drawing its supply from there. The Nova Scotia growers were thoroughly alarmed and willingly undertook any method that promised a way out. In the Church Street district thirteen of the growers with the best seed selected by hand from one-half barrel to fifteen barrels of seed from the best plants in their fields and planted it in 1916 beside seed from the general crop. The result was so far from expectation that it was difficult to explain it on the old theory of the nature of leaf roll, for there was on the average practically as much of the disease in the selected as the unselected seed. For example one grower had 41 per cent of leaf roll in his seed plot while another had 25 per cent.

Although proof was thus given for the first time that the selection of large plants from a crop affected with leaf roll does not eliminate the disease, a further attempt was made in 1916 to improve large areas of seed by rogueing it in the field before it was shipped to Bermuda. The result was again the same, for a high percentage of leaf roll developed in these stocks when they were planted in the South. It was just after these events that the paper of Quanjer et al. (loc. cit.) was received, in which the infectious nature of leaf roll seemed to be proved. Further proof was actually at that time in our hands, for one of the seed-plots just mentioned which had 25 per cent of leaf roll in 1916 showed 40 per cent in 1917. The more exact experiments above recorded definitely established the infectiousness of the disease under Canadian conditions.

## EFFECT OF INTRODUCING NEW SEED

These results ultimately supplied a full explanation of the failure to improve the crop in Nova Scotia by the selection method being used. A way was found in the meantime, however, to remedy the situation, that is, by the wholesale introduction from another district of sufficient healthy seed to replace practically all the old in one season. The new stock gave entire satisfaction for two years, but in the third season 3 per cent of leaf roll was not uncommon. As was to have been expected, some of the growers from lack of faith or excess of curiosity retained a little of their own seed. Thus patches containing 7, 11, 34 and 37 per cent of leaf roll were still found, in some cases beside the new seed. The result of this became finally evident in 1918. One grower after planting his main crop with new seed put in about 1 acre extra with the culls which were left from his old stock. One portion of the latter field developed as much as 13 per cent of leaf roll, while the main field had 1.5 per cent on the average. In the following year the percentage of disease on this farm ran up to 7 per cent although the worst seed was discarded.

Although in the case in which 13 per cent of leaf roll developed the disease tended to be peculiarly prevalent in one low-lying corner of the field, the general evidence with regard to the reappearance of the trouble is not in favor of the theory that infection persisted in the soil. There probably was enough of the old infected seed left to account for the second outbreak. This is in agreement with experience in Bermuda (3).

After the new seed had been grown in the district for four years the situation was again getting out of hand. There was practically no field above suspicion, and steps had to be taken again to circumvent the trouble as before by the general introduction of fresh stock from a practically non-infected district.

### DISEASE-FREE DISTRICTS

The district used as a source of supply of healthy seed is situated only 75 miles from the affected area. The latter is in the fruit section of Nova Scotia, which is the warmest and probably the driest part of the province, surrounding the towns of Wolfville and Kentville. It was formerly believed that the local climate was mainly responsible for the disease situation. It is now doubtful if so much importance can be attached to this factor. In the first place comparatively healthy seed which has never showed more than two per cent of disease, has been maintained on one farm in the affected area without any care other than careful bin-selection. All around this farm from 5 to 50 per cent of leaf roll has been common. The seed is not noticeably resistant, for it has been freely infected both naturally and artificially. According to the record of the grower it has not been changed for many years. In the second place leaf roll has now appeared in a few farms in the previously disease-free district in considerable amounts. There had always been a trace present, generally not more than 1 per cent, but in 1917 up to 6 per cent was found in seed which one grower introduced from an unknown source. Thereafter leaf roll became more common, although all the infection must not be traced back to the case mentioned, for in some instances the growers were found to be raising other diseased varieties in 1919. Five Garnet fields were found containing from 3 to 3.5 per cent in 1918, and in 1919 six fields showed from 3 to 7.5 per cent of leaf roll. It is obvious in this case that what was previously attributed to climate seems to depend more on the restricted sources of infection which were present up until recent years. The conclusion is being reached that while climatic variations of such magnitude as exist between Charlottetown, Bermuda and Ottawa exert a pronounced influence on the relative prevalence of leaf roll, the smaller variations which occur within the Maritime Provinces cannot be held principally responsible for the local distribution of the disease. The extent to which sources of infection are present and the degree to which the disease spreads in the field seem to be dominant factors. The latter depends to some extent on varietal susceptibility, as well as on other factors the nature of which is unknown.

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# A MACROSPORIUM FOOT-ROT OF TOMATO

J. ROSENBAUM

WITH FOUR FIGURES IN THE TEXT

Numerous specimens of tomato plants were received in June and July, 1919, from Delaware, showing a disease the symptoms of which differed markedly from those of any known disease on tomatoes.

*Symptoms.* The symptoms as noted on these plants had a marked resemblance to black leg on potato stems. In figure 1 is shown a photograph of several naturally infected tomato stems, taken at the time these were received. Such stems, at the point where they emerge from the surface of the soil, first turn a dark brown. This brown rot or canker on the stem spreads deep into the tissues and extends above and below the surface of the soil. With age, the infected portion of the stem turns darker, the tissues shrivel up and may break, and the stem falls over at the original point of infection. Occasionally similar brown to black spots are found on other parts of the stem above ground. In such cases it often spreads a considerable distance from the point of infection, involving the entire stem and terminal blossoms.

In many cases the rot at the base of the plants produces a girdling effect. If this occurs when the tomato plants are still young, they throw out new roots above this point. Accompanying the girdling and stem cankers, the plants show varying degrees of wilting and discoloration of the leaves.

The disease has been found on tomato plants of all ages, both in the seed bed and in the field. Where the original infection takes place has not been determined.

The symptoms as outlined above suggest the possible use of "foot-rot" as a common name for this disease on tomatoes.

*Losses.* It is hard to estimate at this time how serious or how widespread the disease may prove to be. In a letter from one of the growers received June 30, 1919, referring to this trouble, he states, "On going through the fields recently fully 40 per cent of the plants were found to be affected with this disease."

*Isolation of causal organism.* Microscopic examination of diseased stems showed fungous mycelium within the affected tissues. No distinctive spores or fruiting bodies were found. Isolations were made on a variety of agar plates. In brief, the procedure followed was to wash the

FIG 1 LESIONS ON TOMATO STEMS DUE TO A MACROSPORIUM

Typical appearance of tomato stems affected with foot rot Natural infection

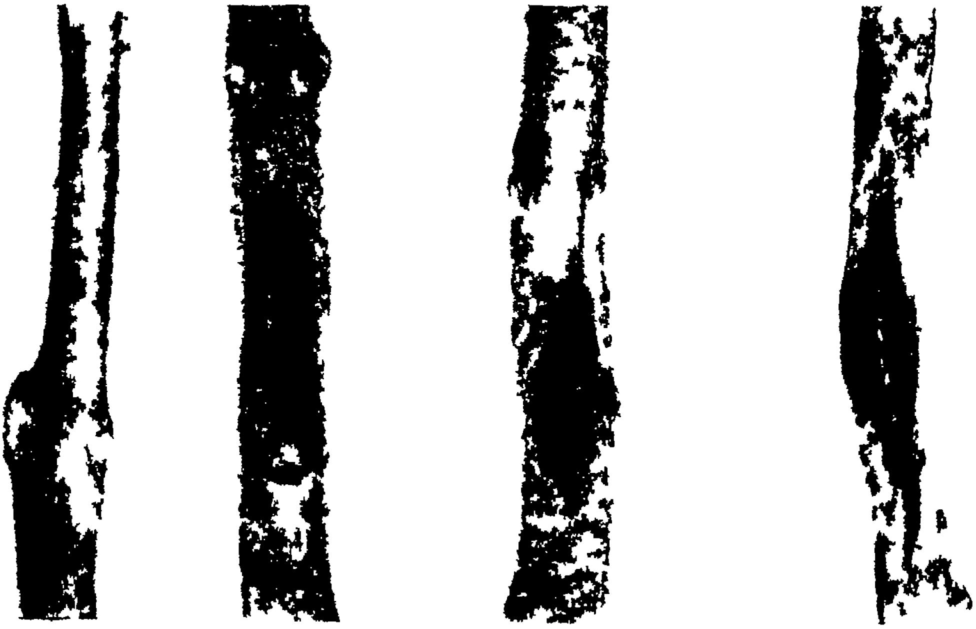

FIG 2 LESIONS ON TOMATO STEMS ABOVE GROUND DUE TO INOCULATION WITH THE FOOT ROT MACROSPORIUM

One check, as shown, remained healthy Photograph taken eight days after inoculation affected stem, immerse in a 1:1000 bichloride of mercury solution for ten minutes, rinse in sterile distilled water, and make plantings from the inner tissues of the affected parts. Dilution plates on beef agar were also made. In the first series of isolations made, 71 per cent of the plantings gave a pure culture of the same fungus. The remainder of the plantings resulted in bacterial growth, or a mixture of bacteria and the same fungus as found in the majority of the colonies. During the progress of this work several series of isolations were made at various times from naturally infected stems. In all of these the same fungus was constantly associated with the lesions.

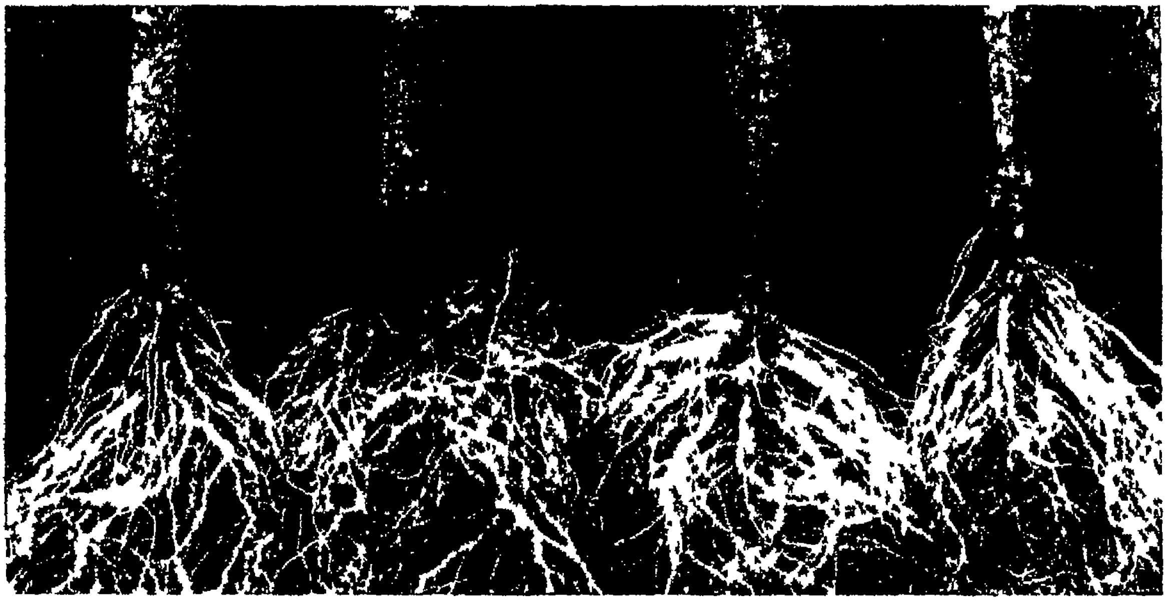

FIG. 3. LESIONS ON TOMATO STEMS BELOW SURFACE OF SOIL DUE TO INOCULATION WITH THE "FOOT-ROT" MACROSPORIUM

One check, as shown, remained healthy. Photograph taken eight days after inoculation.

*Inoculations.* Notwithstanding the predominance of the one fungus obtained from the isolations, it seemed advisable to test in addition, by artificial inoculations, the bacterial cultures obtained. Bouillon cultures of these bacteria were made and inoculated in a variety of ways on injured healthy plants in the greenhouse and in the field. The results in every case were negative.

With the fungus obtained from the isolations, a large number of inoculations were made, both on plants in the greenhouse and in the field. Two varieties of tomatoes were used, viz., Livingston's Globe and Earliana. The inoculations were made on the stems of healthy plants, both above ground and below, and near the surface of the soil. The procedure followed was to cut the stem slightly with a flamed scalpel and insert in the wound a bit of fungus, from a pure culture grown on agar media. The inoculations made below the surface were either covered directly with soil or first covered with a piece of absorbent cotton moistened in sterile water, followed by soil; of the inoculations made above ground, part were left unprotected and the others were likewise covered with a piece of absorbent cotton moistened in water. Check plants were injured and treated in a similar manner, but without inserting the fungus into the cut stem.

The first series of inoculations were made July 3. When these were examined eight days later, the results obtained were so conclusive as to leave little, if any, doubt regarding the cause of this disease.

While the size and the resulting lesions varied in the field and in the greenhouse and on plants of different ages, all showed positive infection. Of the thirty-four plants inoculated, not a single one remained healthy. The checks, on the other hand, in every case remained unaffected. Figures 2 and 3 show the results of part of one series of inoculations. The photographs were taken eight days after the inoculations were made. In each case one check is shown.

*Re-isolations.* Re-isolations were made from a number of these plants. These resulted in a fungus growth similar to the one originally obtained from the naturally infected plants.

*Identity of the causal fungus.* The fungus causing the disease makes a vigorous mycelial growth on a variety of agar media. Neither on the host nor in culture does it produce spores readily. On account of its resemblance to other Macrosporium cultures, it was subjected to the treatment recommended by Kunkel[1] to obtain sporulation with *Macrosporium solani*. The treatment consists of wounding vigorously growing colonies of the fungus mycelium. With this treatment an abundant sporulation was obtained. The spores proved beyond a doubt that the fungus belongs to the genus Macrosporium. In figure 4 are shown camera lucida drawings of several typical spores of this fungus obtained in pure culture, and, for comparison, three other groups of Macrosporium spores. The latter were obtained from cultures of Macrosporium from potato leaves, from typical "nail-head" spots on tomato fruit, and from a rot on tomato fruit from New Jersey.

Examination of the literature failed to reveal any mention of a Macrosporium on tomatoes causing a disease with symptoms such as are described herein. In the Southern and Gulf States, where a Macrosporium on tomatoes causes serious damage and affects the leaves, stems, and fruit, the lesions produced on the stems are small "nail-head" spots, rarely, if ever, penetrating very deep into the tissues of the stem. The symptoms of the known Macrosporium diseases are therefore in marked contrast to those described here.

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[1] Kunkel, L. O. A method of obtaining abundant sporulation in cultures of *Macrosporium solani* E. & M. Brooklyn Bot. Garden Mem. 1: 306–312. 1918.

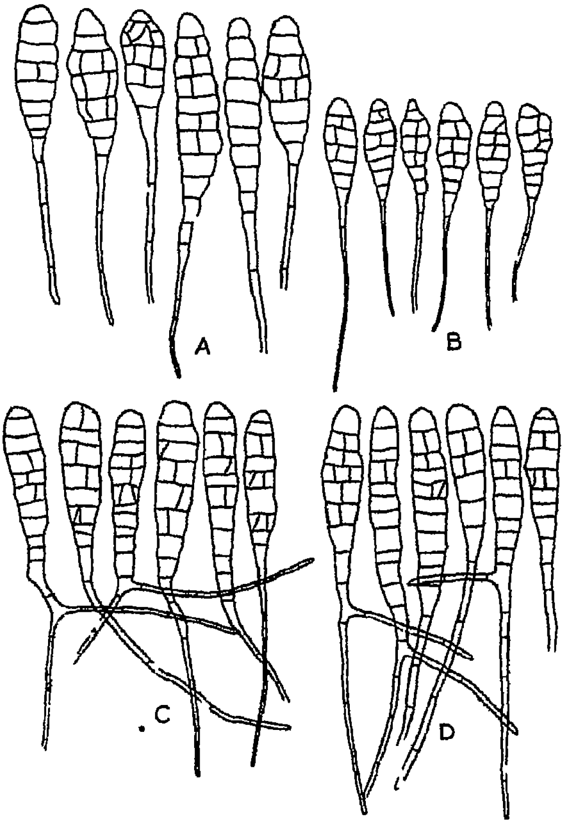

FIG. 4. MACROSPORIUM SPORES FROM TOMATOES AND POTATOES

Spores obtained in pure culture on hard potato agar. A. *M. solani.* Isolated from potato leaves from Maine.

B. *M. tomato.* Isolated from typical "nail-head" spots on tomatoes from Florida.

C. *M. solani.* Isolated from tomato plants affected with "foot rot" from Delaware.

D. *M. solani.* Isolated from potato seedlings affected with damping-off from Maine.

In view of these differences it was of interest to compare the Macrosporium obtained in this case with the others discussed in this paper, as shown in figure 4.

Comparisons[2] were made as regards (a) growth on media, (b) inoculations on various parts of the tomato plant,—stems, leaves, and fruit, and (c) measurements of the spores.

All of the Macrosporiums under discussion were grown on various agar media. The fungus isolated from typical "nail-head" spots could, with ease, be distinguished in each case from the other three Macrosporiums. The latter produced a growth which was characterized by a deep red or claret discoloration of the substratum of the media. The intensity of this color is dependent upon the alkalinity or acidity of the agar. The color is soluble in water and chloroform, but insoluble, or only slightly so, in ethyl alcohol and carbon bisulphide. It exhibits the properties of an indicator, being bright yellow when acidified and red when alkaline. It breaks down upon standing in an alkaline solution in the light.

The Delaware fungus, while not identical in appearance on the various agar media with *Macrosporium solani* from potatoes, certainly resembles

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[2] An account of studies made of Macrosporiums isolated from tomatoes from different sections of the United States was published in Phytopathology 10: 7–22. 1920. In this article the spores marked "D" in figure 1 should be referred to as saprophytic Alternaria instead of a Macrosporium, as the spores are borne in chains.

*Inoculations with Macrosporium*

| SECTION OF PLANT INOCULATED | METHOD OF INOCULATION | CULTURE USED | RESULT OF INOCULATIONS | |
|---|---|---|---|---|
| | | | Infection | Character of lesion produced |
| Stems and leaves | Sprayed with suspensions of spores produced in pure culture | "Nail-head" Macrosporium from tomato fruit | + | "Nail-head" spots in abundance |
| | | Delaware fungus from tomato stems | + | And occasional "nail-head" spot on leaves |
| | | New Jersey Macrosporium from tomato fruit | + | |
| | | *Macrosporium solani* from potato leaves | + | |
| Stems | Incisions made with a flamed scalpel and piece of fungus mycelium inserted in the cut | "Nail-head" Macrosporium from tomato fruit | − | |
| | | Delaware fungus from tomato stems | + | Dark brown rot or canker. Girdling of stems |
| | | New Jersey Macrosporium from tomato fruit | + | Similar to the ones produced by the Delaware fungus, but smaller and not as penetrating |
| | | *Macrosporium solani* from potato leaves | + | |
| Fruit—Various stages of maturity | Sprayed with a suspension of spores produced in pure culture. Fruit not injured | "Nail-head" Macrosporium from tomato fruit | + | Typical "nail-head" spots |
| | | Delaware fungus from tomato stems | − | |
| | | New Jersey Macrosporium from tomato fruit | − | |
| | | *Macrosporium solani* from potato leaves | − | |
| | Incision made with a flamed scalpel and piece of agar with pure growing culture inserted in cut | "Nail-head" Macrosporium from tomato fruit | + | Small, gray-black spot |
| | | Delaware fungus from tomato stems | + | Large, firm, dark brown lesions, extending deep into the tissues |
| | | New Jersey Macrosporium from tomato fruit | + | |
| | | *Macrosporium solani* from potato leaves | + | |

it. It should be stated that while the culture of *M. solani* which was used for comparison in this case produced a discoloration of the substratum, the writer has seen and worked with other cultures of *Macrosporium solani* which apparently do not produce any discoloration whatsoever. The Delaware fungus also agrees in cultural characteristics with the one from New Jersey.

In order to compare the pathogenicity of these Macrosporium cultures and the character of the lesions which they are able to produce, inoculations were made on the stems, leaves, and fruit with each form. A summary of the results is given in table 1. It shows the portion of the plant inoculated, the method of inoculation, and the results obtained in each case. The data presented again points to the fact that the Delaware stem Macrosporium agrees closely with *Macrosporium solani* from potato leaves and with Macrosporium from tomato fruit from New Jersey. It is different from the Macrosporium obtained from typical "nail-head" spots on the tomato fruit, both as regards its pathogenicity and the character of the lesion produced.

TABLE 2

*Spore measurements of Macrosporium*

| CULTURE | SPORE BODY, IN MICRONS | | | | | | BODY OF SPORE, PLUS BEAK, IN MICRONS | | | AVERAGE NUMBER OF SEPTA |
|---|---|---|---|---|---|---|---|---|---|---|
| | Minimum length | Maximum length | Minimum width | Maximum width | Average length | Average width | Minimum length | Maximum length | Average length | |
| Macrosporium from tomato stems (Delaware) | 56.7 | 98.7 | 14 7 | 23.1 | **75 1** | **18.0** | 157.5 | 350.7 | **244.1** | **8** |
| *Macrosporium solani* from potato leaves (Maine) | 29.4 | 111 3 | 8 4 | 23.1 | **67.0** | **18.2** | 71.4 | 247.8 | **153.1** | **7** |
| Macrosporium from "nail-head" spots from tomato fruit (Florida) | 29.4 | 69.3 | 10 5 | 23.1 | **47 3** | **16.9** | 54.6 | 279.3 | **132.6** | **6** |
| Macrosporium from tomato fruit (New Jersey) | 42.0 | 128 2 | 10.5 | 25.2 | **79.0** | **20.1** | 88.2 | 407.6 | **288.5** | **8** |

To determine further the identity of this Macrosporium, measurements of one hundred spores were made from each of the cultures. The spores were obtained in each case from potato agar plates of the same age and grown under the same conditions. A summary of these measurements is given in table 2.

The Macrosporium isolated from the Delaware tomato stems is much larger than the Macrosporium isolated from "nail-head" spots on tomato fruit. The differences in size between the others are not significant. Figure 4 shows the comparative size of the spores from each of these cultures. It also shows that the terminal beak of the Delaware Macrosporium is often forked. This is a frequent characteristic of the spores of *M. solani* from potatoes, as is well illustrated by Rands.[3] Finally, the beak of the spores of the Delaware fungus is coarser than that of the ordinary "nail-head" Macrosporium, but similar to *Macrosporium solani* from potato.

Consideration of the facts brought out in these comparisons leaves on doubt that the Macrosporium isolated from Delaware tomato stems is different from the Macrosporium isolated from "nail-head" spots on tomato fruit, but similar to *M. solani* from potatoes. It should therefore be referred to as *M. solani* E. & M.

## SUMMARY

1. A stem disease of tomato plants was found doing considerable damage in Delaware. The symptoms of the disease suggest the possible use of "foot-rot" as the common name for this trouble.

2. Numerous isolations from infected plants and from artificial inoculations in the field and greenhouse establish the fact that a fungus belonging to the genus Macrosporium is the causal organism.

3. Comparisons of this Macrosporium were made with *M. solani* from potatoes and with Macrosporium cultures isolated from a tomato fruit rot from New Jersey and from typical "nail-head" spots on tomatoes from Florida. These comparisons show that the Macrosporium discussed here differs from the one isolated from typical "nail-head" spots from Florida, in culture and morphology as well as in the fact that the former is unable to produce infection on uninjured green fruit. It resembles *M. solani* and the Macrosporium isolated from tomato fruit from New Jersey, in culture, in the kind of lesions produced when used for inoculation on fruit and stems, and in morphological characters. It should therefore be referred to as *Macrosporium solani* E. & M.

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[3] Rands, R. D. Early blight of potato and related plants. Wis. Agr. Exp. Sta. Bul. 42: 1–48, fig. 4. 1917.

# BRIEFER ARTICLES

## A FROST INJURY OF POTATOES

H. G. MacMillan

With Plate XIII

A peculiar condition of the younger leaves of potato plants during the early growing season has been often noted, for which there has been no satisfactory explanation. The leaves were dotted with small irregular necrotic areas usually less than a millimeter in diameter, the majority of the spots being on the basal half of the leaf, along the midrib and near the petiole. The spots rarely appeared, if ever, after the plant reached fair size and the season was advanced. There was always the possibility that they might have been due to flea-beetle injury, but there was no hole, which would have been characteristic. No reasonable cause could be assigned for the occurrence of these spots.

During February potatoes were planted in pots in the greenhouse for experimental purposes. It was necessary to remove them on April 5, and they were stored in the laboratory basement over night with the intention of placing them out of doors in the sun during the succeeding days. The following morning the plants were set out a few minutes before ten o'clock; the day was warm, the sun bright, and the sky clear. The thermometer registered 39° F. The plants at this time were large, normal, and perfectly healthy in every respect. They had been exposed to no insect attacks or spray mixtures, and had been otherwise carefully handled. On April 16, the newer leaves showed marked symptoms of injury.

The plant shown in plate XIII is characteristic of both the field symptoms and the appearance of the potted plants. The larger leaves which are in focus appear healthy and normal, while the center leaves show the very small necrotic areas. The tips of the larger leaf blades are free from spotting, whereas the bases of the smallest leaf blades are unspotted. The areas are very small, irregular, dead and shrunken. Free-hand sections examined under the microscope showed these areas to be free from parasites, and dried and dead, while the tissue around them appeared healthy and normal. Moreover, many microscopic areas were revealed, not visible heretofore, which involved only a few cells, reaching from the tracheae to the surface through the palisade tissue. Apparently these smaller areas were around stomata, though the cell structure could not be determined on account of the death of the tissue. DeVries, in his treatise on the anatomy of the potato plant, says stomata sometimes occur in the upper epidermis. The larger macroscopic areas extended through the leaf from one epidermis to the other.

The spots had every appearance of being caused by mechanical injury, such as frost. As the circumstances were well known under which the plants were set out on the morning of April 6, the data indicate that injury was caused by frost. The barometer stood at 25.04 inches; the temperature was 39° F.; the day was fair with a light wind from the east. The relative humidity, as recorded by a hygrothermograph calibrated by a sling psychrometer a few days previous, was 45 per cent. According to the Psychrometric Tables of the United States Weather Bureau (W. B. No. 235, p. 73, 1912), a depression of the wet bulb thermometer of 7.5° F. accompanies an air temperature of 39° F. and relative humidity of 45 per cent. This would give a temperature of 31.5° F. for the wet bulb thermometer.

The potato plants had been watered heavily the night before and placed in a warm basement. At that time the now expanded center leaves of plate XIII were very small stipuliform whorled leaflets, the tip ends of the terminal leaflets alone being expanded. All the leaves were turgid, and water probably filled all the stomatal openings. Exposure of the plants in dry air suddenly chilled the moist parts to a freezing temperature, resulting in the frost necrosis, not observable at the time, and only becoming apparent upon the extended growth of the leaves. The environment of the small whorled leaves in a light breeze would be comparable to the wet bulb of the sling psychrometer.

As noted at the beginning, this malady has been observed in the field. During early June warm nights, followed by very sudden cooling in the early morning, often occur. On potatoes a few inches high during the coolest nights these necrotic areas ultimately occurred. The time of appearance lagged so far behind the occurrence that the connection was missed, but in the light of the pot experiment the necrosis of the leaves must have been due to frost injury.

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PLATE XIII

A POTATO PLANT SHOWING FROST INJURY IN THE FORM OF SMALL, IRREGULAR, NECROTIC AREAS ON THE YOUNG, INNER LEAVES

MACMILLAN POTATO FROST INJURY

# DEVICE FOR GROWING LARGE PLANTS IN STERILE MEDIA

J. K. WILSON

WITH ONE FIGURE IN THE TEXT

Many methods have been suggested for growing plants for experimental purposes. These may be divided into two classes: Those which attempt to grow plants under sterile conditions, and those which do not. Of the former methods two have been more or less successful, depending upon the size of the plant to be grown and the duration of the experiment. With small plants, and in experiments of short duration, plants have been grown in suitable containers which have kept the entire plant from infection throughout the experiment. In other methods only that portion of the plant and its surroundings which were of importance in the special investigation have been kept under sterile conditions. In this paper a method is described that is of this latter type.

Investigators have found that it is sometimes necessary to grow plants in a medium from which all other forms of life are excluded, in order that a particular point to be investigated may not be interfered with by some other factor. Since this point usually has to do with the absorption of some particular substance, the contamination of parts other than the absorbing system may not be of any consequence. Edward and Barlow (1) grew the entire plant under sterile conditions. Garman and Didlake (2) started their plants under methods designed for sterile cultures, and as soon as the plants developed, the tops were allowed to grow in the open. Schulow (3) grew plants under a semi-sterile condition and reported good results. The method followed by any investigator is usually developed to meet his particular requirements.

The inability to use satisfactorily in our work any of the methods reported led us to try many ways to remedy this condition.

The most promising method for large plants, such as maize, and one yielding a fair degree of success, is a modification of the method used by Schulow. Briefly, his method consisted in germinating seeds that had been treated with bromine water for twenty minutes. The plantlets resulting were transferred bodily to the mouth of a small-necked container in which was a sterile nutrient solution. They were held in place by sterile cotton, and after becoming sufficiently large were wrapped with sterile cotton and allowed to grow. This kept the roots in sterile surroundings and the tops in the open. The main objections to this method were the difficulty in transferring the plantlets and the great chance for contamination in such procedure. By making certain changes in this method the transferring could be done as satisfactorily as the transfer of an ordinary bacterial culture.

The above method as modified by us is as follows:

### SEED STERILIZATION

Seeds are rendered sterile by the calcium hypochlorite method recommended by Wilson (4). After disinfection the seeds are planted on a sterile medium, from which, after germination, they are transferred to the permanent position. The solid medium for this germination is usually composed of the same ingredients as those used in the large container, from which the plant eventually draws its nutrients, with 1 to 1½ per cent of agar. Agar and distilled water alone may be used, but the former is recommended, so that if contamination is present it may be detected before the plant is transplanted to its permanent position. This medium is made in sufficient quantity to meet all requirements, and is distributed into large test tubes.

Since the roots of most plants spread out in a lateral direction and thus make it difficult to transplant them quickly and conveniently, some method should be used which will direct the root growth in a vertical direction. To accomplish this there is placed in each test tube a short piece of glass tubing, 25 mm. by 50 mm. (see fig. 1, *e*). When the medium is distributed into the tubes, enough is put into each to cover all but about 15 mm. of the tubing. After sterilization of the medium the sterile seeds are dropped into it, where they germinate and produce plantlets for subsequent use. As they grow the roots penetrate the agar medium and pass down to the bottom of the tube. When the roots have thus developed the short piece of glass tubing, with its agar and plantlet, is lifted out and set into the mouth of the prepared flask. This is accomplished with sterile forceps

### PREPARATION OF FLASKS

A flask of any size with a neck that can be properly packed with cotton may be used. As much of the nutrient solution is put into the flask as can be sterilized therein, and a piece of cloth is stretched over the mouth. A hole just large enough to permit the insertion of a piece of 20 to 30 mm. tubing is cut through the cloth (see fig. 1, *c*). This tubing is just large enough to allow the plant and its tube to telescope into it (described above).

The lower end is slightly constricted to prevent the inner tube with the plant from going too far into the flask. Enough cotton is wrapped around the tube to hold it firmly in place when it is pushed into the mouth of the flask. The upper end of the tubing should be about even with the top of the flask. The lower end should extend below the cloth and cotton so that when the flask is full of liquid the latter will just touch the tubing but not the packing. The flask is then covered with a large inverted beaker, which should be deep enough to leave some space above the mouth of the flask for the growing plant. In this way sterility can be maintained without much danger of subsequent infection.

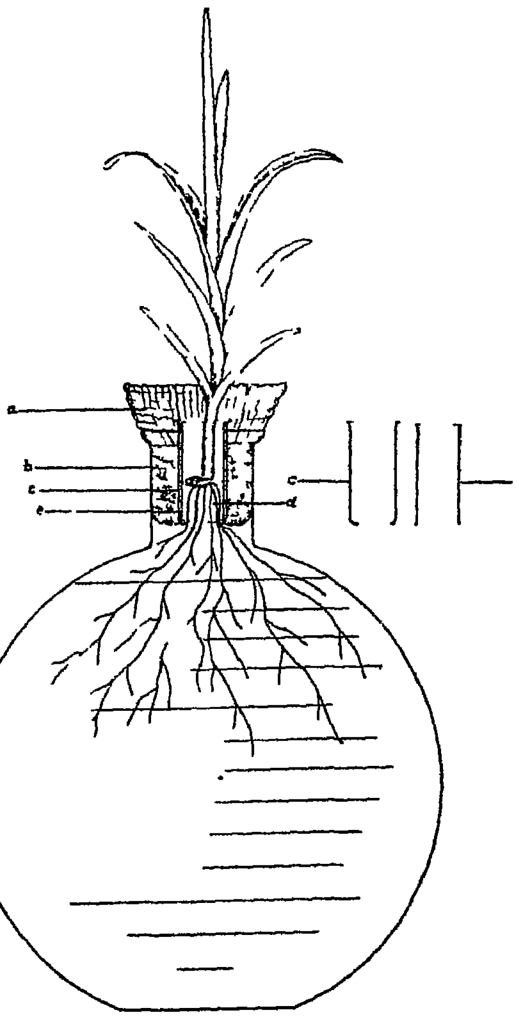

FIG. 1. DEVICE FOR GROWING LARGE PLANTS IN STERILE MEDIA

*a*, Cheese cloth; *b*, cotton wool; *c*, outside tube into which *e* slides; *d*, agar carried over from test tube with corn plant; *e*, tube in which seed germinates.

Since there is need for more solution in the flask than can be sterilized in it, an extra amount is prepared, which may be poured into the flask through the opening that is left. If this opening seems small, a funnel may be sterilized with the flask, and after the latter is filled with the nutrient solution the funnel may be removed.

### SETTING PLANTS

Before a plant is set into the mouth of the flask it should be inspected to note whether there is contamination. Plants transferred from the test tube to the flask need suffer no setback. If the plants are large enough when placed, they may be wrapped with sterile cotton and the beaker removed.

Maize, peas, and oats have been grown to maturity by this method. It should be noted that many kinds of nutrient solutions have been employed. The data presented in the following table were obtained from experiments in which there was no added organic material. The nutrient solution in all cases was of the same composition.

The table presented brings out very clearly what results may be expected from this method. In most cases about one hundred days elapsed before contamination was found. In the last series with maize, where

*Sterility of nutrient solutions when crops were harvested*

| CROP | DATE STERILIZED | NO OF FLASK | | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 1 | | 2 | | 3 | | 4 | | 5 | | 6 | | 7 | | 8 | | 9 | | 10 | | 11 | | 12 | | 13 | | 14 | |
| | | Days to harvest | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | | Days | Sterility | Days | Sterility | Days | Sterility | Days | Sterility | Days | Sterility | Days | Sterility | Days | Sterility | Days | Sterility | Days | Sterility | Days | Sterility | Days | Sterility | Days | Sterility | Days | Sterility | Days | Sterility |
| Maize | June 21 | 28 | + | 43 | + | 56 | + | 70 | − | 98 | − | 85 | − | 91 | − | 98 | − | 108 | − | | | | | | | | | | |
| Oats | October 10 | 37 | + | 60 | + | 80 | ? | 94 | ? | 108 | + | 128 | + | 156 | + | 205 | | 217 | | | | | | | | | | | |
| Oats | February 12 | 43 | + | 78 | + | 99 | + | 99 | + | | | 106 | + | 123 | − | 129 | | 141 | | 150 | − | 150 | − | | | | | | |
| Maize | January 23 | 44 | + | 67 | + | 95 | + | | | 119 | − | 123 | − | 126 | − | 127 | + | | | 170 | − | | | | | | | | |
| Maize | December 18 | 60 | + | 73 | + | 76 | + | 87 | + | 96 | + | 101 | − | 108 | + | 122 | + | 133 | + | 133 | + | 150 | + | 150 | + | 150 | + | | |
| Maize | January 10 | 70 | + | 75 | + | 80 | + | 84 | + | 89 | + | 101 | + | 104 | + | 110 | + | 113 | + | 120 | + | 120 | + | 142 | + | 148 | − | 153 | − |

+ = Sterile.
− = Not sterile.
? = Sterility questionable.

twelve flasks were harvested before the 148th day, no contamination was found. With maize, contamination may occur wherever the brace roots open up the cotton packing.

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## GUMMING DISEASE OF SUGAR CANE IN PORTO RICO

J. MATZ

WITH ONE FIGURE IN THE TEXT

Gumming disease of sugar cane is known to have occurred in Australia (New South Wales and Queensland), Fiji Islands, Mauritius, Java, Borneo, New Guinea, Brazil, and very likely in Tucumán, Argentina. There is no record of it in the West Indies. The disease apparently does not occur in India.

In February, 1920, gumming of sugar cane was identified by the writer in two car loads of cane from the Trujillo Alto district, Porto Rico. The gummy exudation, varying in color from a lemon-yellow to that of honey, at the cut ends of the cane stalk, together with the bright-red vascular bundles in cane in the more advanced stages of the disease, are symptoms that agree with the gumming of cane of Australia as described by Cobb and studied by E. F. Smith. Cultures of the gum were made on potato agar, and the bacterial colonies as well as the transfers of these to potato plugs agree with those of *Bacterium vascularum* described by the last author. Masses of the bacterium in pure culture were smeared on needle-pricked surfaces of leaves of ten healthy, young "Otaheite" canes in an isolated greenhouse. After three months from inoculation all these plants showed, when cut, typical gummy exudation from the fibro-vascular bundles.

Immediately upon the recognition of the fact that gumming disease exists unmistakably in Porto Rico, a special survey was made of all the principal sugar cane growing centers, and it was found that the disease is not wide-spread and that the exact locations of its occurrence are only in the Trujillo Alto district in an area of about fifteen kilometers. Specimens of the diseased cane were shown to men who have planted cane for years but all were unanimous in declaring it a new thing. One planter stated that he saw it to a very limited extent the previous year. The disease occurs in places where no new introductions of seed were made for many years past, and it was found in the oldest varieties grown on the island, namely, "*Otaheite*," "*Rayada*," "*Crystallina*" and "*Cavengerie*" or "*Colorada*." The "Otaheite" is the most severely infected, while the "Cavengerie" is the least infected variety. It is very likely that the disease has been here in insignificant proportions for some time in the past, but the unusually heavy rains at the beginning of the present year have most likely aided it in taking on the form of an epidemic in one district where "Otaheite" is still generally grown. Whether the disease will subside with the advent of normal rainfall remains to be seen. The only safe way to check it is through the planting of resistant varieties. So far the disease has not been observed in the variety "*Yellow Caledonia*." The fact that "*Rayada*" is susceptible makes it rather a difficult problem here, as this variety is very largely grown all over the island. "Cavengerie" shows decided resistance but it is not a very desirable variety here from the millers' point of view.

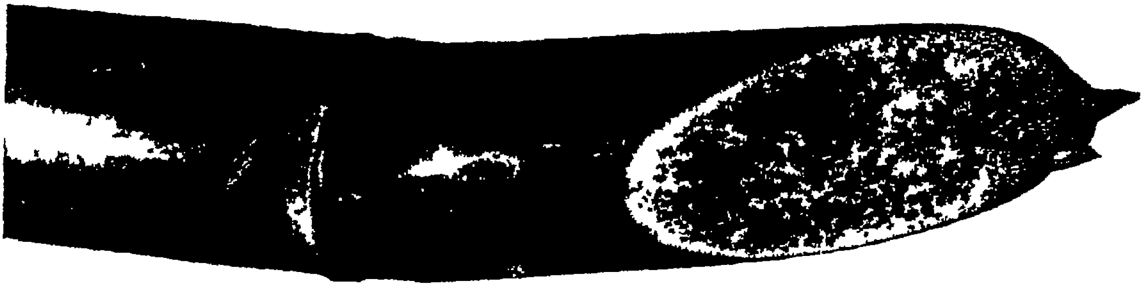

FIG. 1. GUMMING DISEASE OF SUGAR CANE

Gum drops exuding from the cut fibro-vascular bundles of "Otaheite" cane

INSULAR EXPERIMENT STATION
RIO PIEDRAS, PORTO RICO

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Compiled by Alice C. Atwood, and Florence P. Smith, Bibliographical Assistants, Bureau of Plant Industry

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All authors are urged to cooperate in making the list complete by sending their separates and by making corrections and additions, and especially by calling attention to meritorious articles outside of regular journals. Reprints or correspondence should be addressed to Miss E R Oberly, Librarian, Bureau of Plant Industry, U. S. Dept Agric., Washington, D. C

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PHYTOPATHOLOGY

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# WILT OF WHITE CLOVER, DUE TO BRACHYSPORIUM TRIFOLII

LEE BONAR

WITH THREE FIGURES IN THE TEXT

During the summer of 1919 Dr. C. H. Kauffman observed a peculiar diseased condition on a plot of white clover (*Trifolium repens*) on his lawn, at Takoma Park, Washington, D. C. The sod on this lawn was composed almost exclusively of white clover which had been sown on newly cleared land in the spring. The clover seed was bought at a Washington seed store and was used to start the first sod ever grown on this land.

*Symptoms of the disease.* The first noticeable effect of the disease consisted in the decidedly wilted condition of the attacked clover plants. Later all the leaves and petioles of the plants in an infected area became light brown in color, as well as much shriveled and dry. It seemed to appear in spots, which then spread until these coalesced and the whole of the lawn became a withered brown mass. The disease ran most of its course coincident with continued rainy and muggy weather, the last stages of collapse being furthered by the drying out of the soil and exposure to the sun.

Examination of the diseased clover plants at intervals showed them to be infected by a fungus, and some of the fungus was collected and brought to Ann Arbor. In November, 1919, the work of culturing the causal organism was undertaken. Single spores of the fungus were isolated and grown on artificial media. The fungus grew readily in the laboratory and produced the typical conidiophores which had been found on the diseased clover plants in great numbers.

*Inoculation experiments.* Pots of clover were grown in the greenhouse, and when the plants were well developed they were sprinkled with a heavy suspension of the spores of the fungus taken from a pure culture, which in turn came from a single spore. These pots were then immodiately placed under bell jars, in order to prevent drying out. Uninoculated pots were also placed under bell jars to serve as check. Inoculations were made on clover plants varying in development from fifteen days old, with the third leaf just forming, to those that had well developed prostrate stems and many leaves to each plant and were beginning to form flower stalks.

On the sixth day following the inoculation there appeared on several of the leaves of the more mature plants small spots having a water-soaked appearance. These spots were small at first and appeared on a single leaflet. After a few days, however, they were observed to be spreading to the other two leaflets, and then to affect the petiole also. As the infection spread through the leaf, the spot first formed gradually became light brown in color and somewhat shriveled. At the end of ten days after inoculation, a great many of the leaves and petioles showed infection, and as the infection progressed down the petiole the leaves drooped to the ground and became dry and withered. The infection progressed in the petioles as far as the prostrate stems of the plant, but in no case were the stems observed to be infected. After a short time the stems would put out new leaves, which were subsequently attacked by the fungus when the pots were kept under moist conditions. When, however, the pots were allowed the free air of the greenhouse the infection was not so vigorous, and the plant was able to develop or maintain some green leaves. Photographs of pots 1 and 2, figure 3, show the inoculated plants and checks. Pot 1 and the check plants, pot 2, were photographed one month after inoculation and ten days after removing the bell jars and allowing them to grow in the greenhouse air. Attempts to inoculate the prostrate stems by making incisions with a scalpel, introducing the spores and mycelium of the fungus, and then covering the wound with grafting wax to prevent drying out failed to yield any positive results.

*The fungus and the host.* The mycelium is stout, septate, and much branched, and ramifies through the tissues of the clover leaves. Within the leaves it is hyaline to pale brown in color, while the aerial mycelium is dark brown. The invasion of the leaf by the fungus causes such a complete collapse of the tissue that the relation of the mycelium to the host cells is made out with difficulty. In so far as could be determined, the mycelium is, for the most part at least, intercellular and there are no haustoria present.

About the time the leaves begin to turn brown there can be seen appearing over the surface of the leaf the upright unbranched conidiophores. These grow out through the epidermis of the leaf, which is by this time in a shrunken and disorganized condition. The conidiophores are dark brown in color, and measure 60 to 90 by 5 microns. Under the hand lens the growth of these conidiophores and the attached conidia appears as a dark brown, powdery surface in some places on the leaf. The conidia are borne on the tips of the conidiophores, one to several on a single conidiophore, as in figure 1 (*a*). They are brown, 3-septate, and the third cell from the point of attachment is bulged, so that there is a decided hump on one side of the spore, so much so in some cases that the spore is very nearly triangular. For the form of the spore see figure 1 (*b*). On germination the germ tubes are developed from the end cells of the spore (fig. 1 (*c*) ).

Besides the spores above described there was found on the inoculated plants another spore form. Occasional small, dark spots could be seen on the leaves that were in the process of going down under the attack of the fungus. These spots were about 1 mm. in diameter, and under the hand lens showed an apparently lighter margin. Microscopic examination of these spots showed them to be a pushing up of the epidermis by a growth of hyphae from beneath, and showed the lighter margin about the spot to be merely the colorless epidermis which had been pushed up by the growth of the fungus from the underlying leaf tissue. This covering soon burst and disappeared, and exposed an acervulus which stood distinctly above the surface of the leaf tissue. The acervulus is composed of branched conidiophores, which form a very dense layer at their tips. This layer is composed of chains of spores, which are cut off in succession. (See figure 2 (*a*), which is a section 10 microns in thickness.) These spores, so formed, are very different from the above described spores (fig. 2 (*c*)). These acervuli were observed on all the inoculated plants, but soon ran their course, and after a very short time only the large, up-standing conidiophores could be found on the material. Neither of these spore forms were ever found on the check plants, and only the large, septate spore form was ever found in culture.

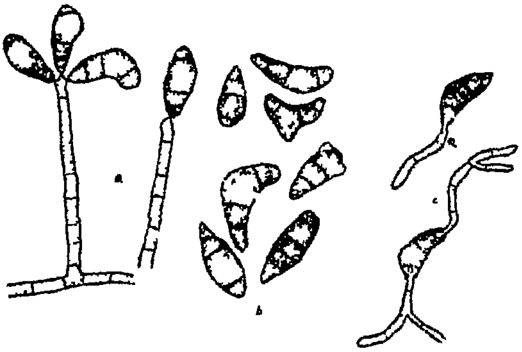

FIG 1. BRACHYSPORIUM TYPE OF SPORES

(*a*) Conidiophores and mode of attachment of spores; (*b*) form of spores; (*c*) germination of spores (21 to 31 by 9 to 11μ).

*The fungus in culture.* The fungus was grown on the following culture media: synthetic sugar-peptone agar, corn meal agar, oat meal agar, potato agar, steamed rice, potato plugs, dead stems of *Melilotus alba*, and on dextrose and maltose solutions. The mycelium is generally dark brown in culture, although there is a wide range of the shades of brown in the different kinds of media used. Spore formation was most abundant on the corn meal and oat meal agars. The greatest number of spores were probably formed on the corn meal agar. On corn meal agar a black felt of aerial mycelium is formed over the surface of the medium, and this is composed of interwoven mycelium and the upright conidiophores. Conidia were also borne on short outgrowths from the horizontal mycelium. These outgrowths were sometimes not more than half the length of the conidia themselves. The agar itself was turned a deep purplish black in color. On oat meal agar, the growth of the aerial mycelium was more marked, as it in some cases practically filled the space under the lid of the petri dish. The medium became at first fawn-colored and then gradually changed to a "deep army brown" (Ridgeway). Potato agar yielded a good vegetative growth, the color changing through a slate-brown to almost jet-black; conidia were very rare in these cultures. On potato plugs the mycelial growth was slow and sparse; no conidia were formed. In maltose solution (2 per cent) there was a growth of blackish mycelium for about ten days, after which growth ceased; no conidia were formed. In dextrose solutions (1 to 5 per cent) the growth was more extensive and continued for one to two months, producing a mat of black mycelium; no conidia were formed. In these solutions irregular clumps of oval to spherical dark-brown cells were developed in the mycelium in great abundance. These were also formed in the various agar mediums. On synthetic-sugar-peptone agar there was a mycelial growth less dense than that on the oat meal and corn meal agars, but there was an abundant formation of conidia on the sugar-peptone agar. On the stems of *Meli-*

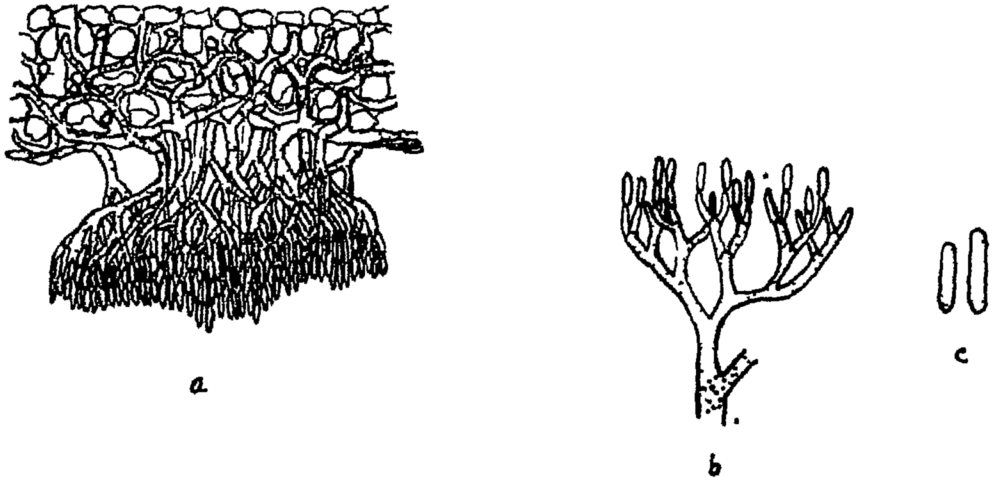

FIG. 2. BLENNORIA TYPE OF SPORES

(*a*) Detailed structure of conidia-bearing hyphae of an acervulus (epidermis has disappeared); (*b*) enlarged portion showing branching and spores in chains; (*c*) single spores. (Section $10\mu$.)

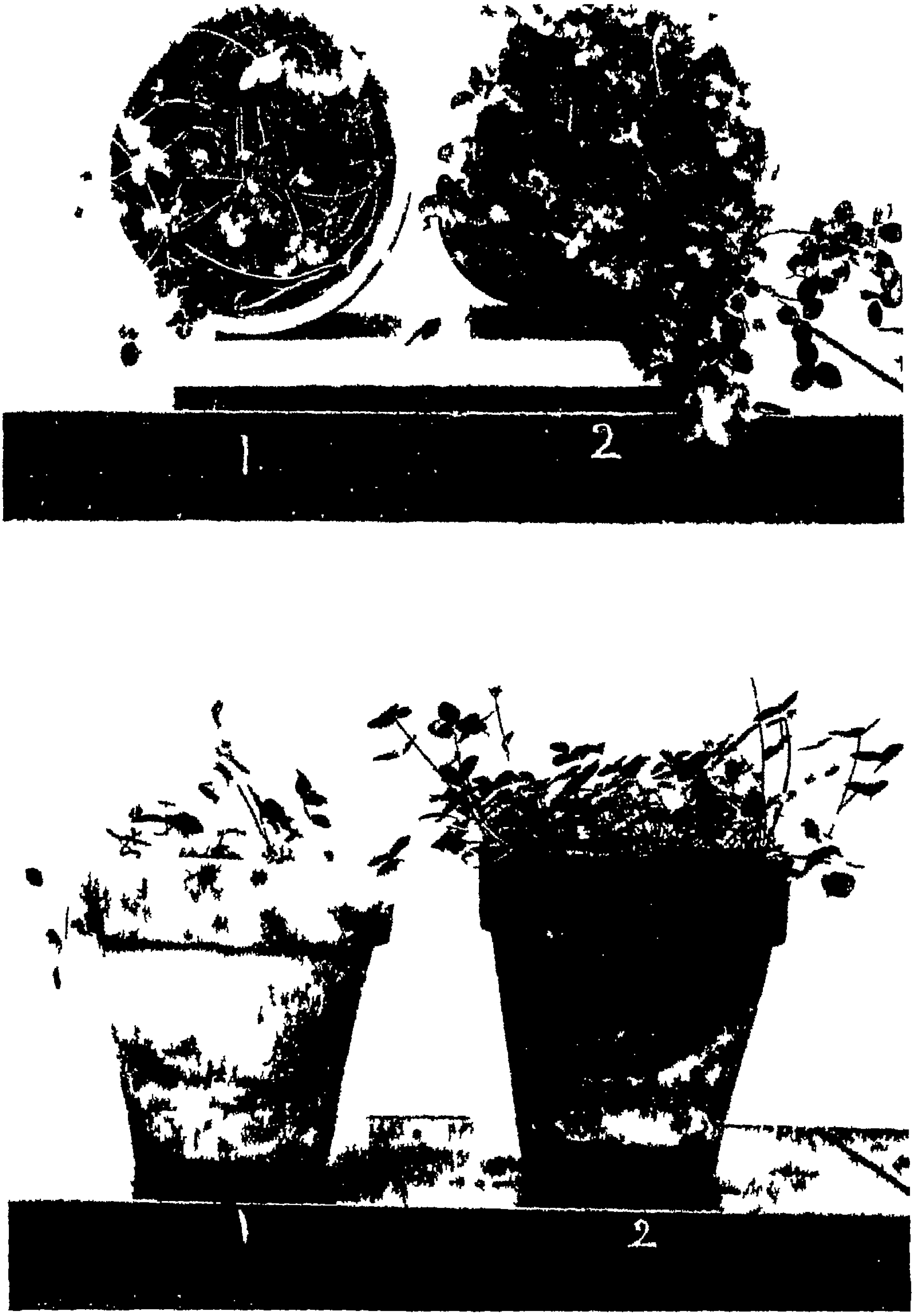

Fig. 3. Pots No. 1 Show Effect of *Brachysporium trifolii* on White Clover One Month After Inoculation. Pots No. 2 Check Plants.

*lotus alba* a dense outgrowth of conidiophores appeared from all sides of the stem, and on the tips of these a very great number of conidiospores were borne.

Cultures of all of the above named kinds of substrata were kept under observation for five months, under varying conditions as to light and temperature. Test-tube cultures were allowed to dry out slowly so that at the end of five months many of them were air dry. No difference of growth was observed between cultures kept in the light and in the dark. No growth occurred in cultures kept at 4° to 6°C. Slow growth, without the formation of conidia, occurred in cultures kept from 10° to 14°C. for seventy-five days. Growth was rapid and conidia formation abundant in cultures kept at temperatures ranging around 20° to 22°C. No growth occurred in cultures kept at 26°C.

*Identity of the fungus.*[1] The larger and very common spore-form clearly belongs to the genus *Brachysporium* Sacc. among the Demataceae. This genus, as at present limited, contains species whose spores, as found in published exsicatti, certainly do not depart much from the typical Helminthosporium spores. The mere fact that they are characterized as shorter than Helminthosporium spores seems scarcely a sufficient generic distinction; at the same time, other species of the genus Brachysporium possess spores approaching or similar to those of the white clover fungus. It would seem that the marked ventricose or asymmetric characters of certain species, along with limited septation, could be made a better basis of distinction between the two genera. Both contain parasitic species, and a more complete knowledge of their life-histories is desirable for mycologic purposes.

The undoubted presence of another spore-form, produced in an acervulus, raises the question as to the proper position of this species. Since no ascus stage has been matured—although suspected—in culture, the naming of the fungus has to do with the spore forms at hand. Theoretically it might be assumed that the acervulus stage is one of higher standing mycologically; still this would be a mere assumption. The characters of the latter spore form throw it into the genus Blennoria Fr. There can be no question, however, that this stage is of secondary importance in the life history of the fungus. This is shown by its sparse occurrence on the host, by its absence in a variety of cultures, and by the fact that infection occurs readily and abundantly when the Brachysporium spores are used exclusively. At any rate such a name would be of little practical value as compared with the one suggested for it. The following is the technical diagnosis of the fungus as far as it is known.

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[1] Identity of the fungus and the technical description by Dr. C. H. Kauffman.

### *Technical description*

**Brachysporium trifolii** sp. nov. Kauffman. Hyphae rigid, brown, varying to sub-hyaline within the host, or dark fuscous in culture, 4 to 5 microns in diameter, septate, branched; dematiaceous conidiophores arising singly, simple, rigid, stout, strict, dark fuscous, 60 to 90 microns long, 5 to 6 microns in diameter; conidia unequally ventricose-pyriform, mostly curved, varying to obovate, very rarely triangular-lobed, obtuse to rounded above, sub-acute downwards, usually 3 septate, antepenultimate cell enlarged, especially on the convex side, dark fuscous brown, thin-walled and smooth, 21 to 31 by 9 to 11 microns. Secondary spore-form in acervuli on the host, sub-epidermal at first, erumpent, conidiophores crowded, much-branched, upwards sub-hyaline, slender; conidia apical in chains, continuous, hyaline, sub-cylindric, obtuse at the ends, 8 by 10 by 2 to 3 microns.

Parasitic on leaflets and petioles of *Trifolium repens*. Takoma Park, near Washington, D. C.

*Conclusions.* As a result of the above recorded studies and a comparison of these with the published records of other clover diseases, it appears clear that we have here a distinct and undescribed fungus which is pathogenic to white clover (*Trifolium repens*).

UNIVERSITY OF MICHIGAN
ANN ARBOR, MICHIGAN
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# OBSERVATIONS ON CYTOSPORA CHRYSOSPERMA IN THE NORTHWEST

ERNEST E. HUBERT

## INTRODUCTION

The weather conditions during the unusually dry summers of 1917, 1918 and 1919 were influential factors in the widespread and abundant appearance of *Cytospora chrysosperma* (Pers.) Fr. upon certain forest, shade and ornamental trees in the Northwest. Observations and experiments have demonstrated that this canker-producing fungus readily enters and parasitizes a host that has previously become weakened or injured or had its twigs or branches killed by drought or other causes. There is, therefore, a real danger of epidemics of this disease resulting from drought.

Long[1] has given a good account of the characteristics of this canker and has drawn special attention to the fact that suppressed, injured or drought-stricken trees are particularly susceptible to the attacks of this fungus.

The fungus is very common throughout the states of Montana, Idaho, Wyoming and Washington, and causes considerable damage to a variety of trees. The following list of collections gives but an indication of its distribution, and includes some new hosts. The numbers refer to the collections in the herbarium at the Laboratory of Forest Pathology, Spokane, Washington. *Cytospora chrysosperma* has been collected and reported from the following places:

On *Populus trichocarpa:* Camp Crook, S. D., No. 6699; Boulder, Mont., Nos. 9204, 9558; Great Falls, Mont., No. 9062; Missoula, Mont., Nos. 647, 3967, 1253; (Propagation cuttings) No. 6696; Twin Lakes, Idaho, reported; Newport, Wash., No. 116; Spokane, Wash., No. 1252.

On *Populus tremuloides:* Deadwood, S. D., No. 11435; Bonner, Mont., reported; Driggs, Ida., No. 87.

On *Populus balsamifera:* Lyman, Wyo., No. 8997; Priest River, Ida., reported.

On *Populus deltoides:* Deadwood, S. D., reported; Missoula, Mont., Nos. 6697, 645; Miles City, Mont., No. 11046.

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[1] Long, W. H. An undescribed canker of poplars and willows caused by Cytospora chrysosperma. Jour. Agr. Research **13**: 331–343, May, 1918.

On *Acer circinatum* (scorched by fire); Missoula, Mont., No. 6697.

On *Acer rubrum:* Spokane, Wash., Nos. 10165, 10568.

On *Acer glabrum:* Locke, Wash., reported; Missoula, Mont., 10029.

On *Prunus demissa* (scorched by fire): Missoula, Mont., No. 6072.

On *Sambucus glauca:* Bellingham, Wash., No. 8053.

On *Sorbus scopulina:* Missoula, Mont., reported, No. 3966.

On *Salix lasiandra:* Idaho City, Ida., No. 6703.

On *Salix spp.:* Wheatland, Wyo., No. 9210; Melrose, Mont., No. 11404; Glasgow, Mont., No. 10566; Darby, Mont., No. 6700; Noxon, Mont.. No. 10763; Addie, Ida, reported; Priest River, Ida., No. 11808; Twin Lakes, Ida., reported; Spokane, Wash., Nos. 10161, 10162.

## SUSCEPTIBILITY OF LOW-VIGOR HOSTS

Inoculations conducted under controlled conditions at a greenhouse at Missoula, Montana, indicate that the fungus is parasitic (wound parasite) when once an entrance is secured through killed or weakened tissues. The following inoculations were made by means of incisions in the bark with a sterile scalpel and spore tendrils from infected material placed within the cut. The inoculated plants were enclosed for five to seven days within celluloid cylinders plugged at both ends with cotton.

### *No. 6682*[3]

Three plants of *Populus trichocarpa* 12 to 15 inches in height were inoculated with pycnospores of *C. chrysosperma* on June 25, 1916. One plant 12 inches in height, with small leaves, was of decidedly low vigor at the time of inoculation; the other two were vigorous (seedlings from natural habitat) as were also the controls. Cankers and small raised pustules were noted on the first plant on September 20, and coiled spore masses were present in abundance on November 25. This plant died. The two vigorous plants failed to become infected. The vigorous plants were seedlings transplanted into the greenhouse the previous season.

### *No. 5987*

Three cuttings of *P. trichocarpa* 16 to 18 inches in height were next inoculated on April 13, 1917. All three plants were noted as dying on October 17. During the month of November a large quantity of pycnospores were produced from typical cankered areas.

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[3] Collection number.

### *No. 5987a*

Three cuttings of *P. trichocarpa* 10 to 12 inches in height were inoculated on April 13, 1917, and then placed for nine days in large test tubes lightly plugged, with water enough to supply each cutting. They were then potted in the greenhouse. Abundant pycnospores were noted on November 16, and more were produced following this date. All three of the cuttings died.

### *No. 6678*

Two plants of *P. trichocarpa*, one 15 inches in height, of low vigor due to drought, and one vigorous 17 inches in height, were inoculated April 13, 1917. The weaker plant developed an abundance of pycnidia by September 5, 1917, while the more vigorous plant developed only one small canker with a few pycnidia in October of the same year.

The inoculation experiments show that the low-vigor plants succumbed to the disease more readily, while the notably vigorous plants did not even become infected, or showed only slight infection.

A large number of field observations corroborate the rather meager inoculation data. On June 21, 1917, a young tree of *Sorbus scopulina* about 12 feet in heigh· was found in a yard at Missoula, Montana, which had the upper half of its limbs and twigs killed by *Cytospora chrysosperma* (No. 3966). Inquiry brought out the fact that drought had caused the first signs of low vigor in the tree. This was soon followed by cankered areas on the upper parts of branches and twigs, and finally death of these parts. The disease finally extended to within three feet of the ground. Careful pruning removed the diseased parts, and new sprouts developed from the base the following year. These remained free of disease during 1918. Several other cases also were noted on the same species of tree, and in all of these low vigor was induced by neglect of the trees. J. R. Weir records a set of three large trees of the above named species in a yard in Missoula, Montana, which received insufficient water during the dry summer of 1917. The trees gave evidence of extreme low vigor. *Cytospora chrysosperma* invaded the dead tips of branches and twigs, and in the fall of the same year all three finally succumbed to the girdling effect of the fungus. He also records a heavy infection of the fungus on the shade trees (*Sorbus scopulina*) at Bozeman, Montana, in 1916. The attack was so severe that a large number of the trees were killed in the same year and were later uprooted and destroyed. Many instances are noted in various cities and towns of the Northwest where cottonwood trees used as shade trees contracted the disease after being weakened by neglect or by injuries and were finally killed by it.

On July 22, 1919, five shade trees of *Acer rubrum* growing along the parking on one of the residential streets of Spokane, Washington, were found in a dead or dying condition. Inspection disclosed the tendrils of *Cytospora chrysosperma* issuing from cankered areas on the lower trunk in each case. Injury to the lower trunk and neglect, were determined as the weakening factors. Very few infections were found in the twigs in this case. Sprouts had developed at the base of the dead trees.

Light ground fires developing in the forest often injure and weaken a large variety of the more hardy shrubs and trees. These later develop characteristic cankers, and finally the deep red spore tendrils of *Cytospora chrysosperma* appear. The host attacked is eventually killed by the fungus. *Prunus demissa, Sorbus scopulina, Acer circinatum, Populus tremuloides, Populus trichocarpa,* and species of *Salix* are the hosts most commonly affected in this manner. No doubt the list of hosts will be extended as collections increase. A collection on *Quercus* sp. (No. 4837) made in Pennsylvania is also to be mentioned here.

On cut-over areas where slash fires develop several years after the logging operations, an enormous amount of the Cytospora disease is found on the weakened and scorched cottonwoods, willows and maple growing on these areas. In these cases both the scorched surfaces of the trunks and the upper parts of twigs and branches become infected. Such conditions hasten the death of the hosts.

The Cytospora canker has been found to be quite common on almost all species of willow, and an unusual form of infection has been collected on this host (No. 10566). In this case a well-formed witches'-broom had developed by the dying out of the main shoots and the subsequent growth of numerous new shoots behind the infected areas. These shoots in time become infected also. Long[3] has mentioned this advance of the disease from the ends of cut-off branches to the new shoots formed about the pollarded areas.

There are two characteristic forms of pycnospore deposits found upon the bark of the hosts. The most common one is the spore-tendril or spore-horn form, in which the pycnospores issue from the pycnidia in long, coiled, dark red masses. These coils are formed under normal moisture conditions. If, however, abnormal moisture is present but not sufficient to wash away the spores, globular, sticky masses of spores are formed about the openings of the pycnidia. These dry down to hard, dark red hemispherical masses. In some few cases a tendency toward lighter colors is found on certain hosts, ranging from a salmon-orange to orange-chrome.[4]

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[3] Loc. cit., p. 332–338.

[4] Ridgeway, Robert. Color standards and color nomenclature. 43 p., 53, col. pl. Washington, D. C., 1912.

## AMOUNT OF DAMAGE CAUSED

The damage caused to trees growing in the forest is vastly greater in amount, though less from a financial standpoint, than that caused to shade and ornamental trees, since most of the hosts attacked in the forest are of the broadleaf species, which are not so extensively used in the lumber industry in this region as are the conifers.

*Cytospora chrysosperma* causes one of the few diseases which are found to damage seriously and eventually kill certain species of shade and ornamental trees. Its habit of attacking trees of low vigor, gradually parasitizing the tissues, and finally killing a portion or all of the tree by girdling, lends itself admirably to the conditions under which a large number of such trees are found. During the dry summer and autumn many of these trees are neglected. Insufficient watering is the most common cause of low vigor, and this results in weakened or killed twigs and branches. Often careless pruning or untreated injuries open a way to the entrance of the fungus. Fire sometimes damages shade and ornamental trees, as well as those in the forest. In nurseries where propagation cuttings of cottonwood and willow are grown the disease is very likely to make its appearance, since under these conditions the fungus readily gains entrance. In the spring of 1917, at Missoula, Montana, about fifty cuttings of *Populus trichocarpa* were planted out in propagating beds by a nurseryman. During the summer every one of the cuttings died. Characteristic cankers were formed near the base of the stems, and upon placing several specimens in damp chambers for one to three days abundant spore tendrils of *Cytospora chrysosperma* were produced (No. 6696). Reports have been received of similar cases in other nurseries. Cuttings of cottonwood shipped into Montana from North Dakota have been found heavily infected by the disease. Care, therefore, should be taken to prevent the spread of the disease through infected nursery stock.

In the Northwestern states, and in Montana particularly, the majority of shade trees planted in the earlier days of the communities were species of Populus. These were selected because they were expected to develop better under the arid and semi-arid conditions of certain localities than some of the other better known or more desirable species. It has been found that the cottonwoods are undesirable for shade trees in parkings, lawns, etc., on account of the damage caused to cement sidewalks and tile piping by the fast-growing and wide-spreading root system. In Missoula, Montana, and in Spokane, Washington, many cement walks have been upheaved and broken by the growth of large roots of cottonwoods beneath them. Cottonwoods are very susceptible to injury through neglect. Lack of water, mechanical injuries and heavy pollarding or pruning being very common forms of neglect and abuse, the trees are easily attacked and killed by the Cytospora. In recent years fewer cottonwoods are being planted, and the old trees are being replaced by more desirable species. The willows, maples and mountain ash (*Sorbus scopulina*) are attacked to a less extent. Few willows are used for shade or ornamentation, but maples and mountain ash are used extensively in this region. The conditions under which the fungus thrives present a real danger of epidemics developing in years of unusual drought.

### CONTROL METHODS

The following control methods are recommended:

*a.* The most resistant species, or those not attacked by the fungus, should be selected for planting.

*b.* Shade trees should be watered regularly and abundantly, and should be protected against mechanical injuries.

*c.* Rigid inspection of nursery stock should be enforced, to prevent the spread of the disease.

*d.* All infected portions of nursery stock and of infected trees found in parkings, lawns, etc., should be cut away and burned.

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# THE EFFECT OF CITRIC ACID ON THE GERMINATION OF THE TELIOSPORES OF PUCCINIA GRAMINIS TRITICI[1]

A. F. THIEL AND FREEMAN WEISS

WITH ONE FIGURE IN THE TEXT

It is generally believed that the teliospores of stem rust will not germinate until spring, following exposure to low temperature and alternate freezing and thawing. Evidence that a natural rest period is required was obtained by failure to secure germination of these spores after subjection to low temperature (−10°C.) and repeated freezing and thawing in early December, 1919.

Means were then sought for shortening the rest period, which it was conceived might be due to either or both of two causes, viz., after-ripening changes in the spore contents or low permeability of the spore wall to water. The following methods of overcoming these conditions were suggested by the work of Crocker (1), Eckerson (3), and Rose (4) on after-ripening in seeds, and of Shull (5) and Denny (2) on the permeability of seed coats.

1. To hasten after-ripening:

*a.* Subjection to increased oxygen pressure.

*b.* Treatment with acids to modify the enzymic changes occurring within the spore protoplasm and to increase its hydration capacity.

2. To modify the permeability of the spore wall:

*a.* Subjection to alternate freezing and thawing and to constant low temperature.

*b.* Treatment with lipoid solvents and acids to change the chemical nature of the wall.

Spores were treated by the methods outlined under 2, but none of the treatments caused them to germinate. However, when placed in dilute citric acid for short periods, they subsequently germinated readily. The apparently specific stimulating effect of this acid on the germination of stem-rust teliospores is considered of sufficient interest to justify a preliminary report.

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[1] Cooperative investigations between the Agricultural Experiment Station of the University of Minnesota and the Office of Cereal Investigations, Bureau of Plant Industry, United States Department of Agriculture.

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On August 12, 1919, telial material of *Puccinia graminis tritici* was collected at University Farm, St. Paul, Minn., and stored outside in wire cages. Short pieces of rusted straw were exposed to chloroform vapor for one minute. It was found that this exposure to chloroform vapor either killed saprophytic fungi or inhibited their growth. Acidulated alcohol was similarly effective but 95 per cent alcohol alone did not prevent the development of contaminating fungi nor did exposure to ether for thirty seconds. In these cases various molds, particularly Penicillium, Helminthosporium, and Fusarium, developed on about one-third of the test material.

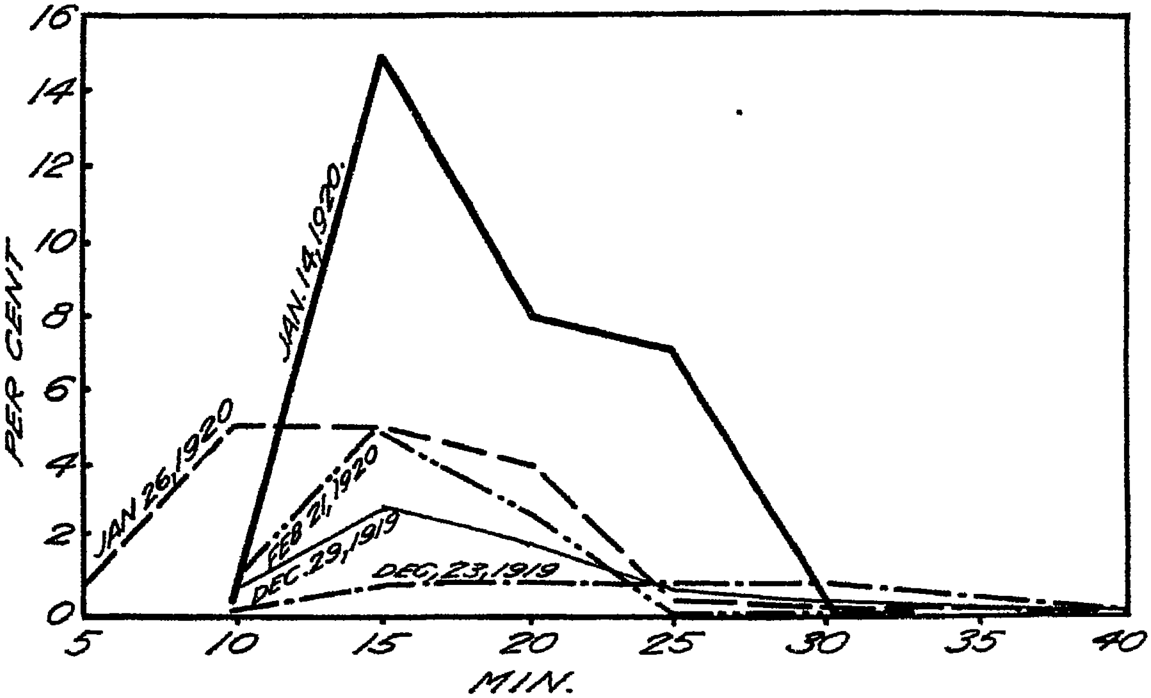

FIG. 1. GRAPHS SHOWING THE PERCENTAGE OF VIABLE TELIOSPORES OF *Puccinia graminis tritici* WHEN TREATED WITH 1 PER CENT CITRIC ACID DURING DIFFERENT PERIODS OF TIME

After exposure to chloroform vapor the stems were immersed for various periods, ranging from five minutes to one hour, in 1 per cent citric acid. The germination tests were then made by placing the treated material in small vials, lined with moist filter paper. These were corked and placed in a refrigerator where the temperature was about 6°C. Experimentation had shown that better germination could be obtained by this method than by any of the hanging-drop methods. The percentage of germination was determined by carefully transferring small portions of the telia to a drop of water on a slide, slightly separating the material, adding a cover slip, and examining under the microscope. This treatment, when carefully carried out, did not result in separation of the promycelia, and subsequent development could be followed on the slide. Counts were made of the number of germinating spores in three different portions of the field.

Teliospores treated with 1 per cent citric acid were examined for germination on December 23 and 29, 1919, and January 14, January 26, and February 22, 1920, as shown in table 1. Control tests were made on each occasion, but no untreated material germinated at any time. The other·treatments listed in table 2 also were not successful in causing germination. The best germination obtained was 15 per cent from teliospores which had been exposed to citric acid for fifteen minutes.

Repeated results indicate that this period of immersion is the optimum (see fig. 1). In one out of five trials 1 per cent of the teliospores germinated after exposure for five minutes and 0.2 per cent germinated after exposure for forty-five minutes. No germination occurred after longer exposure.

TABLE 1

*The effect of citric acid on germination of teliospores of Puccinia graminis tritici. as shown by germination percentages of lots treated for periods varying from five to forty-five minutes*

| DATE ON WHICH EXAMINATION FOR GERMINATION WAS MADE | DURATION OF EXPOSURE IN MINUTES | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 5 | 10 | 15 | 20 | 25 | 30 | 85 | 40 | 45 |
| December 23, 1919 .... | 0 | 0.3 | 1 | 1 | 1 0 | 1.0 | | | 0.2 |
| December 29, 1919 .. | 0 | 0 6 | 3 | 2 | 0 7 | 0.3 | 0.15 | 0 | 0 |
| January 14, 1920...... | 0 | 0 4 | 15 | 8 | 7 0 | 0 2 | 0 | 0 | 0 |
| January 26, 1920....... | 1 | 5 0 | 5 | 4 | 0 5 | 0 1 | 0 | 0 | 0 |
| February 21, 1920..... | 0 | 0 8 | 5 | 3 | 0 2 | 0 | 0 | 0 | 0 |

Germination began after two and a half days. A vigorous, well-developed promycelium with completely formed sporidia was produced in three days. In most cases only the lower cell germinated. The upper cell of a few spores sent out a promycelium, but in no case did both cells of a single spore germinate.

On February 25, 1920, teliospores which had been exposed to citric acid for fifteen minutes were placed on leaves of *Berberis vulgaris* and *fremontii* and the plants were then kept in a moist chamber for forty-eight hours. The beginning of pycnial development was clearly visible on both hosts on March 7, and abundant pycnia were formed by March 10. On February 27 inoculations again were made, some with teliospores which had been treated with citric acid and others with spores which had been soaked only in distilled water. Pycnia again appeared in eleven days where treated spores had been used, but no infection was obtained from the checks.

Tests also were made with butyric acid, as this has been shown (3) to be effective in hastening after-ripening of seeds, but inconclusive results were obtained. Germination of a single spore was observed in material that had been stored indoors since its collection in November and not subjected to alternate freezing and thawing. On the other hand, no germination was obtained from material which had been stored outside all winter. Further experiments are under way.

It is highly probable that germination of teliospores might have been obtained much earlier had this treatment been discovered earlier in the season. The nature of the effect of citric acid is at present under investigation, and various salts of the acid are being tested. The fact that lipoid solvents and sodium hydroxide were ineffective, though ordinarily increasing the permeability of cell walls to water, makes it appear doubtful that the effect is wholly on the spore wall. The fact that acids other than citric do not produce a similar effect may indicate that the process is not one of hydrogen ion catalysis. There is at least a possibility that the acid may act as a specific activator, as it is known that the lipase of castor-oil seed requires activation by an acid, although in this case any acid will suffice.

TABLE 2

*Treatments used in germination experiments with teliospores of Puccinia graminis tritici, the combination employed being indicated by asterisks. No germination was obtained in any case*

| CHEMICAL | TREATMENT | | | | | | TREATED MATERIAL EXPOSED TO | | |
|---|---|---|---|---|---|---|---|---|---|
| | Soaked in distilled water one hour placed in solution for number of minutes indicated. | | | | | | Alternate freezing and thawing | Temperature of 6°C in ice box. | Temperature of 25°C in incubator |
| | 10 | 20 | 30 | 40 | 50 | 60 | | | |
| HCL, $\frac{M}{100}$ | * | | * | | | | * | * | * |
| Phosphoric acid, 1 per cent | * | | * | | | * | * | * | * |
| $H_2SO_4$, $\frac{M}{100}$, $\frac{5M}{100}$, $\frac{M}{10}$, and $\frac{2M}{10}$ | * | * | * | * | * | * | | * | * |
| Tannic acid, 1, 2, and 5 per cent | * | * | * | * | * | * | | * | * |
| Lactic acid, 1, 2.5 and 5 per cent | * | * | * | * | * | * | | * | * |
| KCL, $\frac{M}{100}$, $\frac{5M}{100}$, $\frac{M}{10}$, and $\frac{2M}{10}$ | * | * | * | * | * | * | | * | * |
| Phenol, 1, 2.5 and 5 per cent | * | * | * | * | * | * | | * | * |
| $H_2O_2$ (Merck's), 0.0625, 0.125, 2.5, and 5 per cent | * | * | * | * | * | * | | * | * |
| Acetic acid, 1, 2.5 and 5 per cent | * | * | * | * | * | * | | * | * |

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## BRIEFER ARTICLE

### APPLE BLACK ROT (SPHAEROPSIS MALORUM BERK.) IN OREGON[1]

C. R. STILLINGER

While some investigational work was being carried on in the orchards about Corvallis, Oregon, during the fall of 1916, a number of apples with the larger part of their surfaces blackened were noticed hanging upon the trees. In one orchard four large trees of a yellow variety, but apparently seedlings, were found with the greater part of the fruit affected in this way. In fact, both the branches of the trees and the ground underneath were covered with plump as well as shriveled black apples. The general appearance of the affected specimens was so suggestive of black rot that an examination was made for cankers or leaf spots, but none were found.[2] Further search for the disease on apples kept in storage by the Horticultural Department of the Oregon Agricultural College revealed the fact that it was quite common upon the stored apples. The trouble was found upon Northern Spy, Rhode Island Greening, Wagner, Delaware Red, Baldwin, and Newton apples.

Since up to this time the typical black rot of the East had never been reported definitely from the Pacific Coast, an investigation of this disease was made. Previously, Lawrence (1) had reported from western Washington a black spot canker on apple trees, which, although he considered it a possible black rot canker, proved to be the northwestern apple tree anthracnose. Dr. F. D. Heald of the Washington State Agricultural College, Pullman, Washington, in a personal conversation, stated that he had received some apple bark from Vancouver, Washington, during the fall of 1918, infected with what appeared to be black rot, but that he had not been able to investigate the case further.

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[1] This work was done in the laboratories of the Botany and Pathology Department of the Oregon Agricultural College, Corvallis, Oregon. The writer wishes to thank Prof. H. P. Barss for his many helpful suggestions and the facilities afforded for carying on the work.

[2] Mr. S. M. Zeller, Department of Pathology, Oregon Agricultural College, Corvallis, Oregon, in a letter dated September 2, 1920, writes that he has collected apple leaf spots this summer at Corvallis, Oregon, and Salem, Oregon, which produce a rot on apples very similar to that produced by a culture of *Physalospora cydonae* secured from Hesler.

In 1915, in an unpublished thesis for the Pathology Department of the University of California, Hahn (2) reported an investigation of an organism that fits the description of the eastern black rot in practically every detail. Because the fungus produced more two-celled than one-celled pycnospores, he finally called it a Diplodia, but could assign it to no particular species. However, the later work of Hesler (3), in 1916, showed that it was not at all unusual for a strain of black rot to produce a majority of two-celled pycnospores. He says: "It appears that certain species of Sphaeropsis have been confused with those of Diplodia. The two genera are separated on the basis of one-celled spores in the former and two-celled spores in the latter. But both genera fail in their chief distinction, so that mycologists have been misled on this point. . . . ." "Apparently, from a single pycnidium in which the spores are of a given type there may develop in succeeding generations a wide variation in size, color, shape, and septation. In an early stage the Macrophoma type may appear in both size and color; later the Eu-Sphaeropsis type, one-celled, brown; and finally the Diplodia forms. The variable shapes that may be found in the generations succeeding a given type indicate that two-celled forms may be mere deviations in the life cycle. . . . ."

"Cultural studies were made of an apple strain (no. 82) with reference to this and other morphological characters. A single ellipsoidal, brown, one-celled pycnospore, 10.8 by 21 $\mu$ in size, from a pycnidium in which all the spores were one-celled . . . . was isolated on March 3, 1913, and a pure culture was developed from it. Examination of the culture at intervals showed the development of hyaline, Macrophoma-like spores. At the end of twenty-four days these had become brownish, and, while the majority were ellipsoidal, averaging from 9 to 10 $\mu$ by 21 to 22 $\mu$, a few were pyriform and measured 12.6 by 30.6 $\mu$. At the end of fifty days an occasional spore was found with a cross-wall; after eighty days 30 per cent of the spores were two-celled, and on May 27, 60 per cent were bicellular."

Thus Hesler's experiments showed that the same strain passes through a stage of development from a one-celled, hyaline-greenish to a septate, brownish spore, the type of the spore depending upon its age. Hahn noticed this in his California strain, and based his decision on the older type of spore. Regarding the rot, Hahn states, as a result of his comparative study of the California fungus with a New York and Massachusetts strain of black rot: "This rot is identical with that caused by the Eastern Sphaeropsis, both in appearance and aggressiveness." However, he considered the dark, two-celled, elliptical spore the mature type, and therefore called the fungus a Diplodia. Thus it is very probable that Hahn found the black rot organism in California, but did not recognize it from what was known of the life history of the eastern blackrot at that time.

The strain occurring in Oregon is very probably different from that found by Hahn, since his fungus produces a canker on apple trees, while the Oregon fungus results in a rot of the fruit. The characteristic appearance—the coal black color—makes the Oregon strain easily recognizable wherever the disease may occur. Infected fruit will generally remain longer upon the tree than ripened fruit, even hanging there over winter. In the early stage of the disease, although the apple may be completely infected by the fungus, the fruit remains quite firm and leathery, retaining the original plump appearance of the normal apple. Gradually, however, the apple shrivels and dries into a hard, wrinkled mass, due to the breaking of the cuticle by the fruiting bodies, which permits the rapid evaporation of the moisture from the apple. The surface of the apple turns black up to within a short distance of the advancing margin of the spot invaded by the fungus. Yet, there is generally an area of about 5 mm. bordering the blackened area, which is brownish. The above mentioned fruiting bodies (pycnidia) are scattered quite generally over all the older portion of the blackened surface of the apple.

Internally the fleshy portion of the apple is of a lighter color than the external surface, being brownish black. Here also appears a distinct brown layer separating the healthy from the diseased tissue, which seems to be quite as firm as the healthy portion. The fungus spreads uniformly in all directions in the apple, showing no particular preference for any region. The mycelium penetrates the tissue in the intercellular spaces. It accumulates directly under the cuticle of the apple as a dense, black mass, which gradually enlarges and apparently dissolves its way through the cuticle. At the same time the cells are crushed below, and thus a pocket is formed, filled with a pseudo-paranchymatous mass of hyphae, which gives the structure a reticulate appearance.

As the fungus grows, this pseudo-parenchymatous mass becomes less distinct, and pycnidia develop, which finally rupture the cuticle. These pycnidia are either unilocular or multilocular, and accordingly vary a great deal in size; they sometimes occur singly, but usually in groups of two or three. Inside the pycnidium is a mass of spores held together by a mucous-like substance. This oozes forth in a curled, white, tendril-like cirrhus, which may be 5 mm. long. These spore horns are very conspicuous as they coil out over the surface of the apple. Microscopic examination shows that the mass of spores is held together by a gelatinous substance, which gradually dissolves in water and liberates the spores. After water washes away the spores, a black body remains, appearing very much like a perithecium, with a round ostiolate opening projecting out of the cuticle, the fragmented edges of which may be seen partially inclosing the structure. Conidiophores are found in the pycnidia. In some cases they consist of single, long, unbranched filaments bearing a spore from the side of each cell; occasionally there is more than one spore produced per cell. In other cases the conidiophores are branched and spores may be scattered along the filaments. In all cases they are sessile and are not aggregated. On the other hand, the spores that occur in the cirrhus are oval to elliptical; hyaline to greenish; generally unicellular, some septate; measuring on an average 9 microns wide by 25 microns in length.

The fungus has been grown upon potato-dextrose agar, Czapek's medium solidified with agar, Czapek's medium as a liquid, and crushed apple tissue. In all of these culture media it grew rapidly and abundantly. The mycelium is appressed to the medium and is hyaline in young cultures, but in older ones it becomes greyish and the substratum black. Thus the fungus produces the same discoloration in culture media that it does in the tissue of the apple. Distinct zonation occurs, the outer zone being hyaline to greyish, the inner and older zones turning greyish black. The optimum temperature for growth is about 20° to 25°C., the fungus being greatly retarded in growth at 15° and killed at 35°C.

After about two weeks, either on solid or liquid media (if the culture is not disturbed), the mycelium collects on the surface in little sclerotia-like masses, which form pycnidia. These develop successively from the center of the culture outward, and are more uniform in size than those on the apple in nature. Eventually a yellowish drop oozes from each pycnidium, resting as a globular body on the top of it. This globular body is composed of thousands of spores held together by a gelatinous substance. The manner of exudation of the spores is somewhat different from that previously described as occurring from the pycnidia on the apple fruit under natural outdoor conditions; it does develop, however, upon inoculated apples that have been kept in a damp chamber. The spore horns that occur in the field have also not been observed in culture. They are evidently reduced to this globular mass, due to the greater amount of moisture present in the air in the cultures. No spores have been found except those that occur in this gelatinous mass or in the spore horns.

The fungus was also grown upon Baldwin and Newton apples. The apples were sterilized with alcohol, washed in sterile water, and placed in a damp chamber. A small section of each apple was raised with a sterilized scalpel and a few spores from a globule of a pycnidium developed in culture were inserted into the apple. In three days the inoculated spots began to turn brown; in eleven days pycnidia were forming upon the apples; and in fourteen days globular exudations began to appear. Within a month the apples were completely blackened.

To determine whether the fungus would infect without a wound through which it might gain entrance, Baldwin and Newton apples were sterilized in 95 per cent alcohol for five minutes, then washed in sterilized water and placed in a damp chamber. Drops of water and drops of Czapek's medium, both sterilized, were placed upon the skins of seven apples, and spores of the fungus from a pure culture were introduced into the drops. Some drops on three other apples were left uninoculated. In five days browning of the spots occurred; in seven, slight blackening was evident; and in twelve, typical pycnidia appeared through the surface of the apples. In all but one case, where the water had either dried up or run off, infection was secured and the typical disease produced. The checks showed no indication of infection. Thus direct penetration by this strain is possible if a small amount of water is present to allow germination. Although Hesler was unable to infect unbroken tissues of the apple fruit with the strains with which he was working, yet this strain seems to be able to do so. Burrill and Blair (4) suggested that this might be possible, while both Scott and Rorer (5) and Brooks and DeMeritt (6) actually demonstrated the penetration of healthy leaf tissue by black rot.

Puncture inoculations upon apple branches, brought into the laboratory during January and placed in large bottles containing water, failed to result in any effect upon the branch other than the injury due to the puncture at the point of inoculation. The inoculated places were covered with celloiden tubes with the ends filled with cotton, one end of the cotton being in contact with water so that the inclosed air in the collar was kept moist. Under these conditions the branches started growth and produced leaves. No inoculation experiments have as yet been made in the field.

To test the viability of the spores, some of the shriveled apples were placed outside in a box near the window, where they would be subject to winter conditions. A few spores that would germinate were found in the pycnidia in the spring.

Since this fungus seemed to agree so well with the published descriptions of the eastern black rot, it was compared with two of the eastern strains in culture and on apples. One was received from Doctor Hesler of Cornell and one from the Bureau of Plant Industry, United States Department of Agriculture. Under the same conditions in tube cultures and plate cultures on the same media no differences could be noted except that this particular Oregon fungus grew somewhat faster, produced a little denser mycelium, and fruited more quickly.

Comparative inoculation studies on Baldwin and Newton apples showed a difference only in rate of growth, there being no variation in the rot produced. The pycnospores produced by the Oregon form were a trifle smaller and had a lighter color than the two eastern strains, which varied somewhat between themselves. Hahn also found considerable variation in the two eastern strains with which he worked, one from New York and one from Massachusetts.

Thus this paper reports for the first time the black rot, *Sphaeropsis malorum*, from Oregon as well as the very probable occurrence of this disease in Washington and California. In none of these states does the disease appear to be of great economic importance as far as observed. The necessity for a more extensive study of the strains of Sphaeropsis and Diplodia has been very much impressed upon the writer as a result of this investigation and he hopes soon to carry on a study of this nature.

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# PHYTOPATHOLOGICAL NOTES

*Personal.* A department of plant pathology has been established at the University of Arizona. Professor J. G. Brown, instructor and assistant professor in the department of biology since 1909, has been appointed head of the new department and plant pathologist of the Agricultural Experiment Station.

Mr. Koichiro Kawakami, assistant in plant pathology of the Botanical Institute of Tokyo University, Japan, succumbed to pneumonia on January 15 of this year. Kawakami's work is for the most part yet unpublished. His work was in the main on the problems of citrus canker, in which he showed great perseverance, patience in obtaining conclusive results, and ingenuity in devising experimental methods. His unpublished work is at present being prepared for presentation by Dr. Tyozaburo Tanaka, formerly of the Bureau of Plant Industry, United States Department of Agriculture, and now residing in Japan.

Kawakami was twenty-nine years old at the time of his death. His education was in the elementary schools of his birthplace, Kisarazu. Chiba prefecture, and in the schools of Tokyo. After graduation from the University of Tokyo he was a fellow of the graduate school of the University where he was fortunate in working under Professor Shirai, chief of the plant pathology division. He later became assistant in plant pathology of the same university. His personality was unusually pleasing, and his interests were deep and sincere. He was an admirer of our American universities and research institutions, and frequently expressed the hope that he would be able to visit the United States for further graduate studies. His death is not only a loss to his friends, his university, and his profession in his own country but is also a loss to the progress of our profession in America and other countries.

H. Atherton Lee,
*Philippine Bureau of Science.*

# LITERATURE ON PLANT DISEASES[1]

COMPILED BY ALICE C. ATWOOD, AND FLORENCE P. SMITH, BIBLIOGRAPHICAL ASSISTANTS, BUREAU OF PLANT INDUSTRY

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[PHYTOPATHOLOGY for September (**10**: 407–434 plate XIII) was issued September 22, 1920.]

# PHYTOPATHOLOGY

VOLUME X NUMBER 11

NOVEMBER, 1920

# COLLAR-ROT OF APPLE TREES IN THE YAKIMA VALLEY

J. W. Hotson

With 15 Figures in the Text

## INTRODUCTION

The Yakima Valley is one of the principal fruit-growing regions of the State of Washington. During the years 1913–1916, fire-blight became so prevalent that it threatened the very existence of the apple industry in the Valley. By drastic action this disease, although not exterminated, is being partially controlled. At the present time (1920) the orchardist is facing another type of trouble that, largely on account of the subtleness of its behavior, promises in the long run to be almost, if not quite, as serious a menace as the fire-blight. A general survey of the orchards from Selah to Grandview revealed the prevalence and seriousness of diseases attacking fruit trees at or near the crown and generally spoken of as "collar-rot" or "crown-rot."

The present article has to do with this malady as it occurs on fruit trees in the Yakima Valley, where most of the investigation was carried on, although some experiments were made on trees in the vicinity of Seattle.

## LITERATURE

The literature on this subject has been quite fully reviewed by Grossenbacher (18).[1] That particular phase of the subject relating to arsenical poisoning is also reviewed by Swingle and Morris (37). Various writers working on similar types of injury have attributed the trouble to a variety of causes. In Illinois (4), New York (18, 35), Wyoming (25), Nebraska (11–12), and Connecticut (6) it has been reported to be due to winter injury or frost, while in West Virginia (16), Pennsylvania (26), and certain parts of New York (38) it has been definitely associated with the fire-blight organism *Bacillus amylovorus*. In Colorado it has been attributed to excessive irrigation (28), woolly aphis (28), and arsenical poisoning (23, 24, 39). *Clitocybe parasitica* has been shown to be the causal organism in Oklahoma (40), *Xylaria polymorpha* in Virginia (13, 14, 15) and North Carolina (41), while *Armillaria mellea* has been found associated with a similar condition in various parts of the United States. Other fungi like *Polystictus versicolor*, *Thelephora galactina* (31), *Sphaeropsis* (18, 27) and *Pleurotus* (18) have been occasionally found associated with collar rot cankers.

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[1] Reference is made by number to "Literature cited," p. 484.

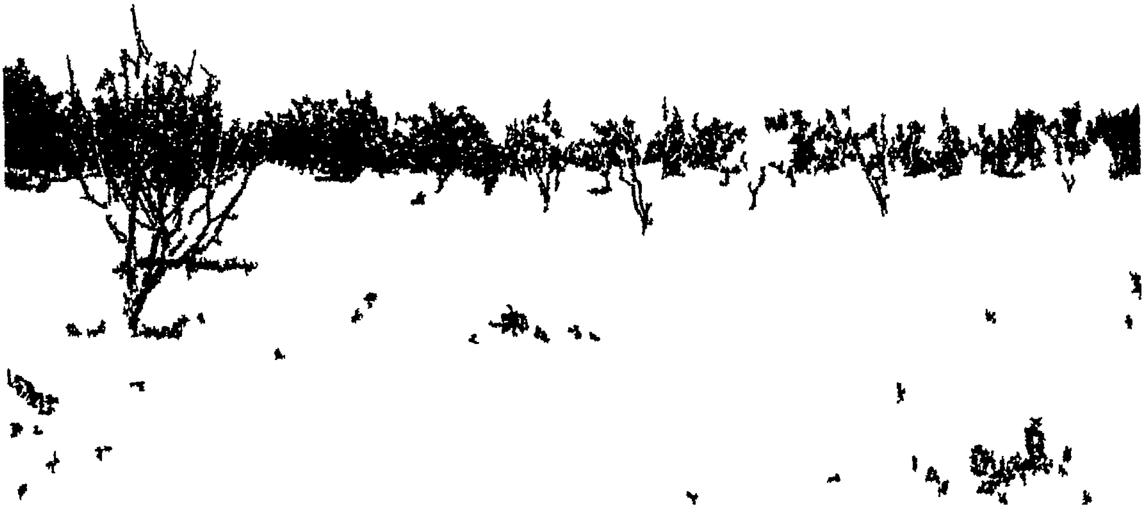


FIG. 1. AN ORCHARD SHOWING APPLE TREES IN THE ADVANCED STAGES OF COLLAR ROT

Those in the foreground are already dead.

### EXTENT AND DISTRIBUTION

If collar rot means the presence of a canker at or near the crown of a fruit tree from whatever cause, then it would appear from the above that this disease is quite widely distributed over the United States. In the Yakima Valley it is considered extremely serious. The examination of a certain 5-acre orchard near the city of Yakima showed twenty-five trees in which the disease could be readily detected simply by the foliage. By a careful tree to tree inspection, removing the soil from the base of each tree to a sufficient depth to determine whether it was infected, over one hundred trees were found showing various stages of development of the disease. An orchard more seriously affected than the one just mentioned is shown in figure 1, where from the point from which the photograph was taken at least thirty affected trees could be recognized, those in the foreground having already died. An examination of other orchards emphasized the fact that, although only a few trees might show the effect of the disease on the foliage, a much larger number showing the canker without the foliage symptoms could invariably be found by making a careful survey.

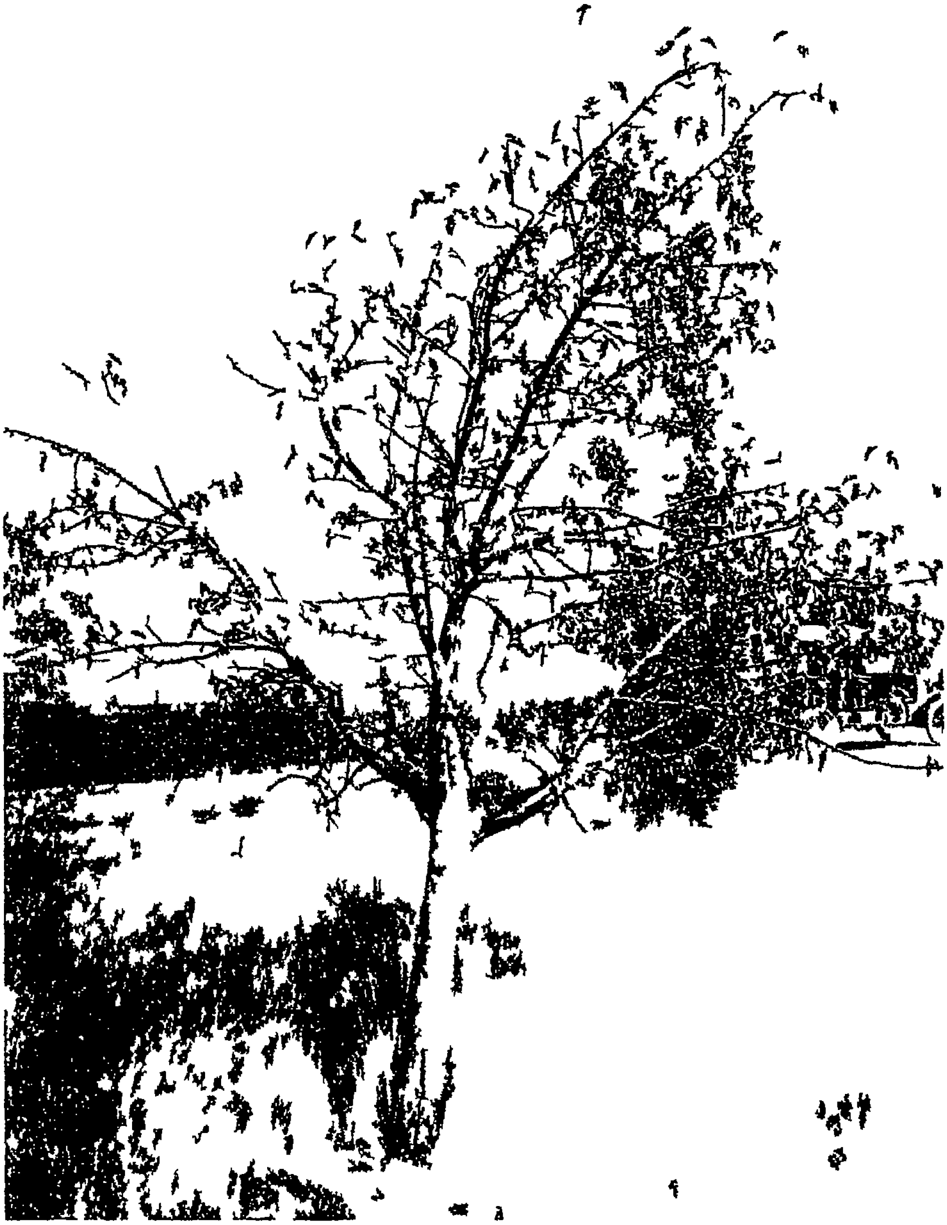


FIG. 2.—A TREE AFFECTED WITH COLLAR ROT

Showing a general sickly appearance with small leaves sparingly developed

## SYMPTOMS

The most striking symptoms of collar-rot are seen during the growing season. The leaves become yellowish and much smaller than normal ones. In the earlier stages of discoloration of the foliage, a single limb or portion of it may show signs of the disease. This limb usually appears on that side of the tree on which the canker occurs; at least it lies along that portion of the vascular system that passes through the cankered area. As the disease progresses the whole tree takes on a sickly appearance, and the limbs, especially on the south side, become a light yellowish or yellowish-red color and more or less scalded in appearance. This condition of the bark may be due in part to malnutrition, but it is mainly due to the excessive action of the sun, since there is only a limited amount of foliage.

These general symptoms are sufficient for the detection of a tree in the advanced stages of the disease nearly as far as it can be seen. One of these trees is shown in figure 2, and one of the cankers in figure 3.

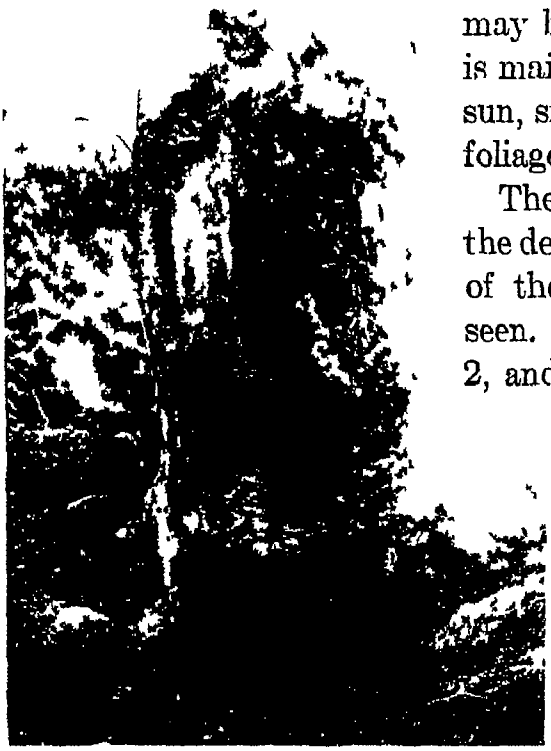


FIG. 3. A TYPE OF COLLAR-ROT WHERE THE INITIAL INJURY WAS AT THE CAMBIUM OR PHLOEM

An examination of the crown of such a tree reveals a more or less conspicuous sunken area. This canker varies in size according to the length of time the disease has been working and the character of the original wound. As the bark is killed it appears brownish, and in advanced stages frequently becomes almost completely transformed into a chocolate-colored powder. In some instances the outer portion of the bark is left intact, while the inner part becomes more or less disintegrated. The disease gradually extends downward into the roots, less rapidly upward in the trunk, and very slowly laterally. The wood beneath the affected bark often becomes water-soaked, brownish in color, and frequently charred in appearance. This discoloration eventually extends into the heart of the stem and root.

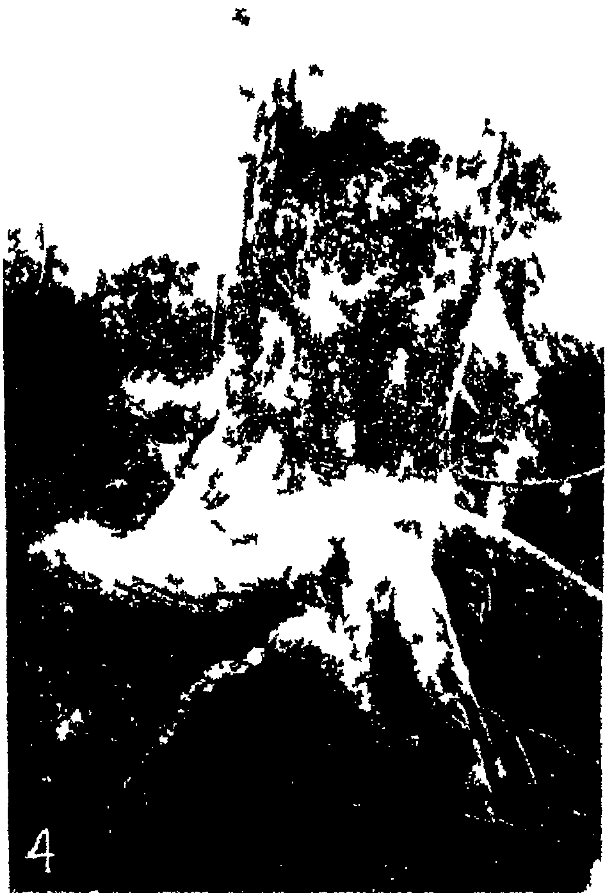
One of the serious features of this disease is the fact that, since the trouble begins under ground or near the surface, it is overlooked by the orchardist until some evidence is seen in the foliage, the whole tree taking on a sickly appearance. Unfortunately, at this stage the disease has frequently become so far advanced that no effective remedy can be applied.

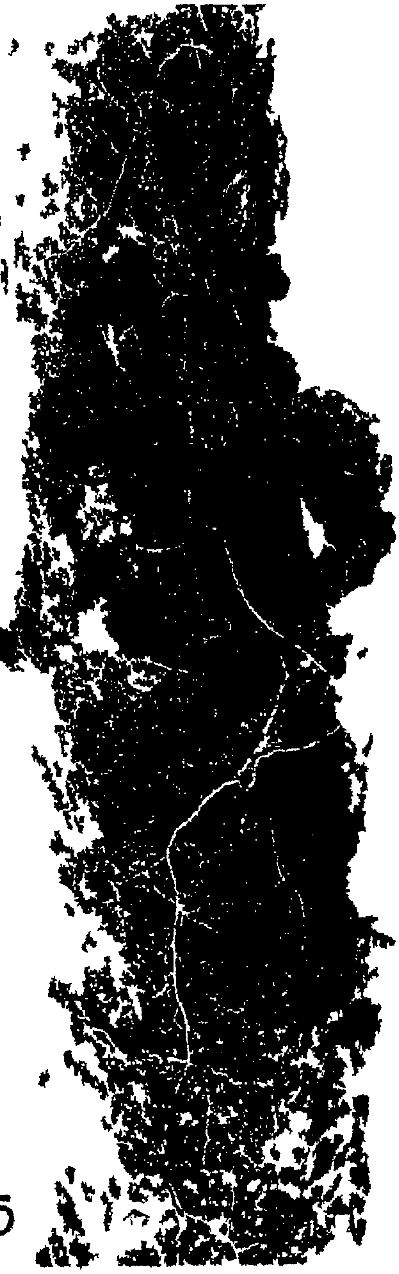
### ORGANISMS ASSOCIATED WITH COLLAR-ROT

When a wound fails to heal an opportunity is given for a variety of organisms, either parasitic or saprophytic, to become established and eventually to gain entrance to the center of the tree. In the Yakima Valley definite organisms, such as *Armillaria mellea, Polystictus versicolor* and *Bacillus amylovorus,* have been found associated with these cankers, *B. amylovorus* having been shown to be the primary cause of some of them. *A. mellea* was found definitely associated with at least twenty different apple trees, and reports were received of its occurrence on many others. In some instances the rhizomorphs had but recently invaded wounds of long standing, while in other cases they seemed to have become established shortly after the wound was formed, and in still others they almost completely covered the roots without any apparent infection. This fungus was identified by the typical, shining, grayish-black rhizomorphs found mostly just below the bark (fig. 5).

It is recognized that the presence of these rhizomorphs is not an absolute proof that the organism is *A. mellea,* but a comparison of the cultural characters of some of these grown on potato agar with those of a known pure culture leaves little doubt that the rhizomorphs found in connection with diseased trees belonged to this organism. In a few instances after the trees had died sporophores were found, but in the majority of cases, the only means of identification was the rhizomorphs.

*Polystictus versicolor* was found occasionally on apple and prune trees, especially in old orchards. It is not limited to its attack on the roots. but occasionally, like *A. mellea,* it may gain entrance to the tree at a wound near the crown, and from this infected area the mycelium may spread upwards and downwards, producing heart-rot. A prune tree attacked by *P. versicolor,* which is producing sporophores, is shown in figure 7. This tree was first observed in the spring of 1914 with its bark peeled off just at the ground, leaving an irregular wound about the size of one's hand. A superficial examination showed no fungus although the tree may have been infected at that time. During the summer of 1915 the tree began to show signs of root trouble, in the leaves, and by middle of August it was dead. That fall sporophores began to form, and in the following spring when this photograph was taken they were abundant.





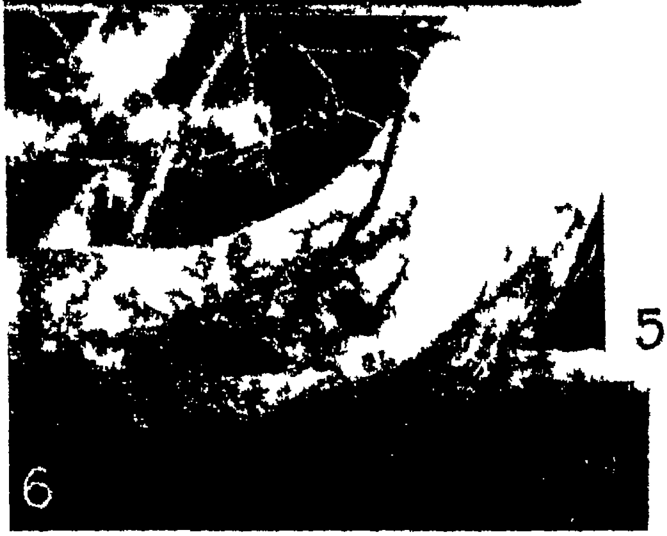


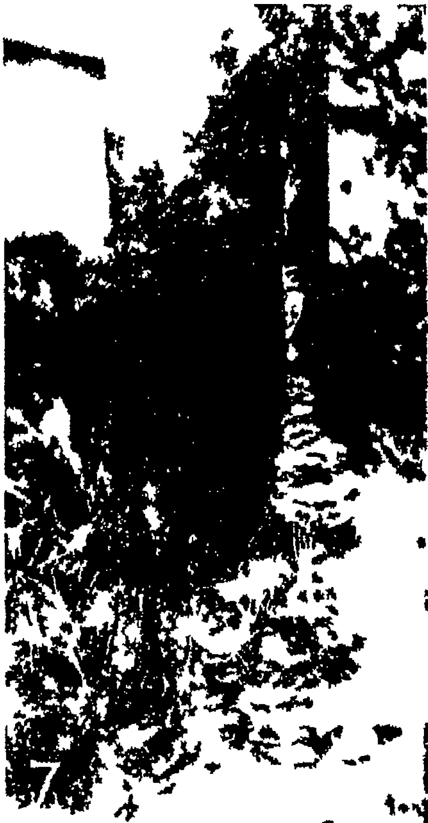
FIG. 4. A TYPE OF COLLAR ROT PRODUCED BY A PROCESS OF CORROSION FROM THE OUTER SURFACE OF THE BARK

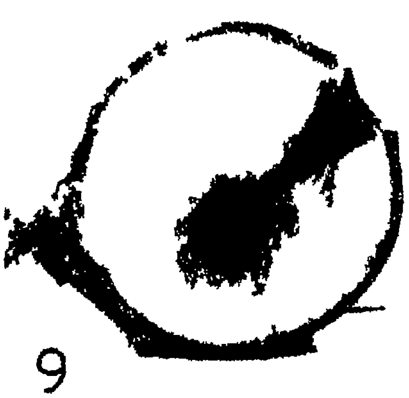
FIG. 5. RHIZOMORPHS OF ARMILLARIA MELLEA ON THE ROOT OF AN APPLE TREE

FIG. 6. A YOUNG CANKER PRODUCED BY BACILLUS AMYLOVORUS

A, a watersprout affected with fire blight and giving rise to the canker

Not infrequently cankers produced by the fire blight organism (*Bacillus amylovorus*) were found at the crown of trees or on the roots near it As is well known the work of this organism may continue in the inner bark until it has girdled and killed the tree or after a period of more or less activity these bacteria may die leaving a sunken cankerous area





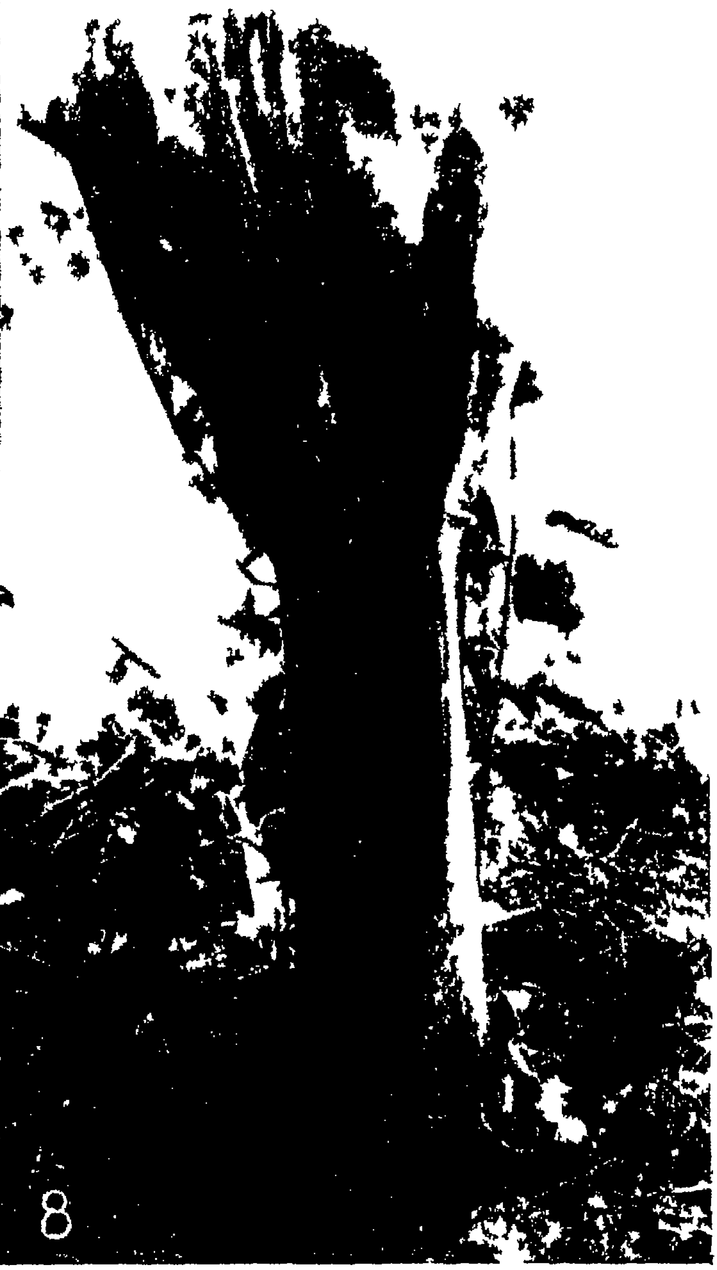


FIG 7 POLYSTICTUS VERSICOLOR GROWING ON A PRUNE TREE

FIG 8 FROST INJURY

The bark on the south side of the tree is peeled off and new cambium growing over practically the whole wound

FIG 9 A CROSS SECTION THROUGH A BRANCH KILLED BY ARSENATE OF LEAD

which develops into a distinct collar-rot condition, and, being difficult to trace to its primary cause, this is usually placed in the same category with cankers of unknown origin.

An instance of blight collar-rot is seen in figure 2, where an attempt to rescue the tree has been made by bridge grafting. The beginnings of a typical collar-rot are shown in figure 6, where a blighted water sprout (a) has been traced back to the root, forming a small canker. The bark has completely decayed over a small area, about 3.5 cm. by 10 cm., with no very definite line of demarcation between the sound and diseased tissue. Cultures on green pears from this diseased bark produced *B. amylovorus*. The wood below the bark was found to be discolored just the same as other types of collar-rot.

It is probable that the large majority of cankers found at the crown of fruit trees in Yakima Valley is due to fire-blight. This statement is difficult to prove after the organisms are dead and there is little opportunity of connecting the two except by comparison with cankers that are known to be produced by the fire-blight organism. An attempt was made to do this. Watersprouts infected with fire-blight were traced to the root and the cankers produced were carefully examined. Some of these were marked and left for several months, and re-examined. Cankers of unknown origin found on trees throughout the valley were compared with these. The result of this comparison seemed to show conclusively that many of the cankers that were previously considered of unknown origin were in reality inactive fire-blight cankers. Many of these evidently had been inactive for some time, probably several years. During this time irrigation and seepage water, which frequently contains a relatively high percentage of alkali, soaked into the wounds, penetrated into the heart-wood and discolored it, possibly by some chemical action set up by the minerals in the water or by some oxidizing enzyme.

## NON-PARASITIC CONDITION

While definite organisms were found in connection with some forms of collar-rot yet more often none could be obtained, although repeated search was made for them. Most of these cases, as has just been explained, were apparently inactive fire-blight cankers. Some, however, were undoubtedly the result of other agencies. In at least two orchards the wounds at the crown of the trees were due to the work of gophers. In several others in which trees showing the characteristic foliage of collar-rot were found, an examination showed that the hardpan was only one-quarter to one-half a meter below the surface, and that the water failed to drain off readily, in some cases resulting in the roots being actually surrounded by water for a considerable time after irrigation, often not draining off before it was time to irrigate again; while in other instances, perhaps only a few rods away, the water seeped away normally and the trees being in such shallow soil, soon began to show drouth conditions. In cases where the water failed to drain away the young roots were killed and soon became water-soaked, turning soft in texture and brown in color. Although the effect of this trouble on the foliage is practically the same as the collar-rot, it is more properly called a "root-rot," as the trouble usually begins at the smaller roots and gradually works back, sometimes along a single root, in which case it might reach the crown before any evidence of it is shown in the leaves. In such cases it resembles the typical collar-rot and may be mistaken for it, but more frequently several roots are affected and the tree is killed before the disease reaches the crown.

Winter injury and carelessness in cultivation are common sources of injury to fruit trees at or near the crown. That frost frequently produces such injuries has been generally recognized for many years, having been reported by a number of writers (4, 5, 10, 11, 12, 25, 27, 35, 39). These wounds, if slight may heal, but if for some reason they fail to do so, the irrigation and seepage water soaks into the wood often producing a brownish stain. The amount of discoloration is partly due to the age of the wound, which may be several years old, and partly to the amount of alkali and other minerals contained in the water that comes in contact with the wound.

Since winter injuries apparently begin at the inner phloem or cambium, the destruction of either of these causes the bark associated with it to die and begin to disintegrate. If these injuries occur at the crown they may undoubtedly give rise to a collar-rot, but winter injuries are far from being the only cause of this malady. In fact, in the Yakima Valley comparatively few of these cankers can be traced to this cause. In a certain orchard west of the city of Yakima and also one in the Naches Valley beginnings of collar-rot could be traced quite definitely to winter injury. Some of these wounds that were wrapped with pieces of cotton or old gunny sacks so as to protect the cambium from the excessive heat during the summer healed over almost completely in one season. This is shown in figure 8, in which the young cambium can be detected covering almost the whole wounded surface. These orchards, both in alfalfa, were inadvertently irrigated so late in the fall that the water had not all drained off before the frosts came. This excessive amount of water freezing in the bark near the ground caused it to split, in some cases forcing it from the wood and producing frost cankers. As the soil offered some protection to the roots these cankers did not extend more than 25 cm. below the surface. Most of the lesions, however, occurred higher up on the trunks of the trees, producing typical frost injuries.

In some cases the primary cause of the wound leading to collar-rot has been traced to defective grafting. An examination of a number of young trees from three to ten years old, killed by collar-rot, warrants the conclusion that defective unions of grafts are responsible for wounds that are the fore-runners of collar-rot.

## ARSENICAL POISONING

Not infrequently there were found cankers that did not seem to be produced by any one of the conditions already mentioned. The final results were practically the same as those of winter injury or fire-blight, but the initial stages and the general effect on the bark were quite different. The resemblance of some of these cankers of unknown origin to those produced by an "ant exterminator," which was found to contain arsenic, and which when applied to the trunks of apple trees injured some and killed others, led the writer to run some experiments to determine something of the effect of arsenate of lead on growing fruit trees.[2]

In the following experiments both branches and roots were treated. The arsenate of lead was obtained from the tank of the orchardist as he was spraying. This spray consisted of an acid arsenate of lead in the form of paste with a strength of four pounds to 100 gallons of water.

### *Effect of arsenate of lead on branches*

For convenience branches that were nearly horizontal were selected for these experiments. In some cases the bark was slightly punctured with the point of a scalpel, deep enough to reach the cambium, while in others the arsenic was applied to lenticels and in still others to the uninjured bark.

The method of treatment was practically the same in all three cases. Where a wound was made a Van Tiegham cell was fastened over it and sealed with grafting wax, care being taken not to allow any of the wax to get into the puncture. The cell, which held about 2 cc., was nearly filled with the solution of arsenate of lead and a cover glass was cemented on the top of the cell with vaseline. In four days a strip of dead bark about 2 cm. wide was found, extending from the point of puncture to the tip of the branch, which in some instances was over 8 feet long, and passing out into lateral branches, if there happened to be any in its path. The leaves along this path were killed but others on the same branch were uninjured. The branch, however, was not completely dead, for the next year the leaves came out as usual. A cross section through one of these limbs is shown in figure 9. The discolored area forms a wedge-shaped structure running into the center, the sides of which are very straight and apparently follow medullary rays. An examination of the margin of the wound shows how the new growth is attempting to heal it over.

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[2] A review of the literature on arsenical poisoning of fruit trees is given by Swingle and Morris (34).

In most cases where Van Tiegham cells were arranged over lenticels a small discolored area appeared around each. In some few instances no change could be detected, probably due to the inability of the solution to penetrate the tissue. In all cases the action seemed to be completed by the end of three or four weeks, and by the following year a decided tendency to heal over the injury was observed.

When Van Tiegham cells were placed over areas on branches where there were no lenticels nor wounds no detrimental effect on the bark was observed. These observations are practically the same as described by Swingle and Morris (37). Quite different results were obtained when the arsenic was applied to the roots.

### *Effect of arsenate of lead on roots*

Lead arsenate obtained from the same source as that used in treating branches was applied to the roots. In these cases the soil was removed and a portion of the root as near the crown as possible was selected for treatment. The applications were made to the uninjured surface and to wounds. Both the solution and paste were applied so that the root would have the irritant action, if any, of the undissolved material as well as the dissolved. The wounds were made by cutting off small rootlets or by puncturing the bark into the cambium in a manner similar to that done in connection with the branches. Van Tiegham cells were used here at first but it was found more convenient to wrap the treated wound with several layers of cotton cloth. After the arsenate had been applied freely and the wound wrapped, the covering was well moistened with water and the soil replaced. In some instances a second application of the arsenate was made but in the majority of cases the initial treatment was the only one. Once a week for six weeks, less frequently after that, these trees were visited, the soil and bandages removed and any differences noted. In each case after the cotton covering was replaced it was well saturated with water so that the treated area was not allowed to become dry. In these examinations the chief changes observed were the increasing size of the cankers and the continued disintegration of the bark. In three weeks a definite action could be noticed where the wounds were made. The bark began to turn brownish about the wound, the discoloration increasing more rapidly longitudinally than laterally. Twelve months after the wounds were made a typical collar-rot canker with the brown, friable bark and deeply stained wood had been developed. Three roots treated in this way are shown in figure 10. These were taken from the trees twelve months after they were first treated. An enlarged view is shown in figure 11 and cross sections of the same roots in figure 12. In specimen "C" the bark is the most markedly disintegrated and is reduced to a more or less powdery brown mass. The disease seemed to have worked faster in this case than in the others, possibly because the tree was in lower and more alkaline soil. In a few instances where the wound was allowed to dry and become aerated it gradually healed, even with the dried white arsenate paste still adhering to it, but where there was plenty of moisture with alfalfa to aid in keeping the humidity fairly constant the cankers gradually increased in size and eventually became permanent.

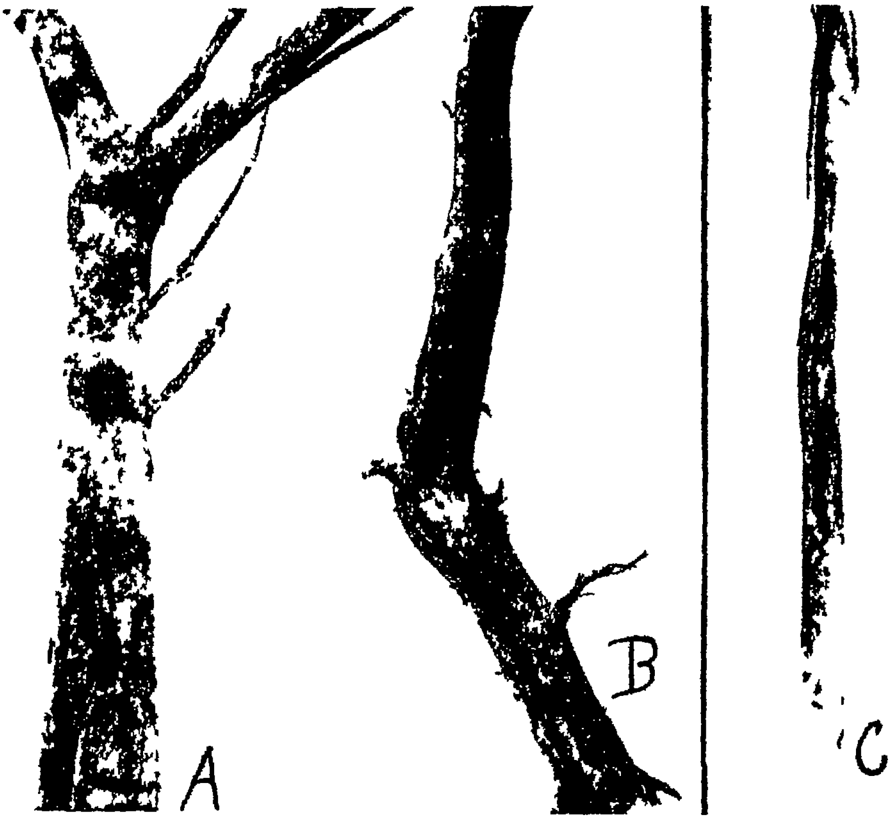

FIG. 10. THREE ROOTS SHOWING CANKERS PRODUCED BY ARSENATE OF LEAD

It is thus seen that the effect of arsenate of lead on wounded roots that are kept moist is markedly different from that on the stem, where it is almost impossible to keep treated wounds constantly in a saturated atmosphere. This is somewhat different from the conclusion arrived at by Swingle and Morris (37), who found that in Montana there was little difference in the effect on the stem and root, and "that excessive moisture

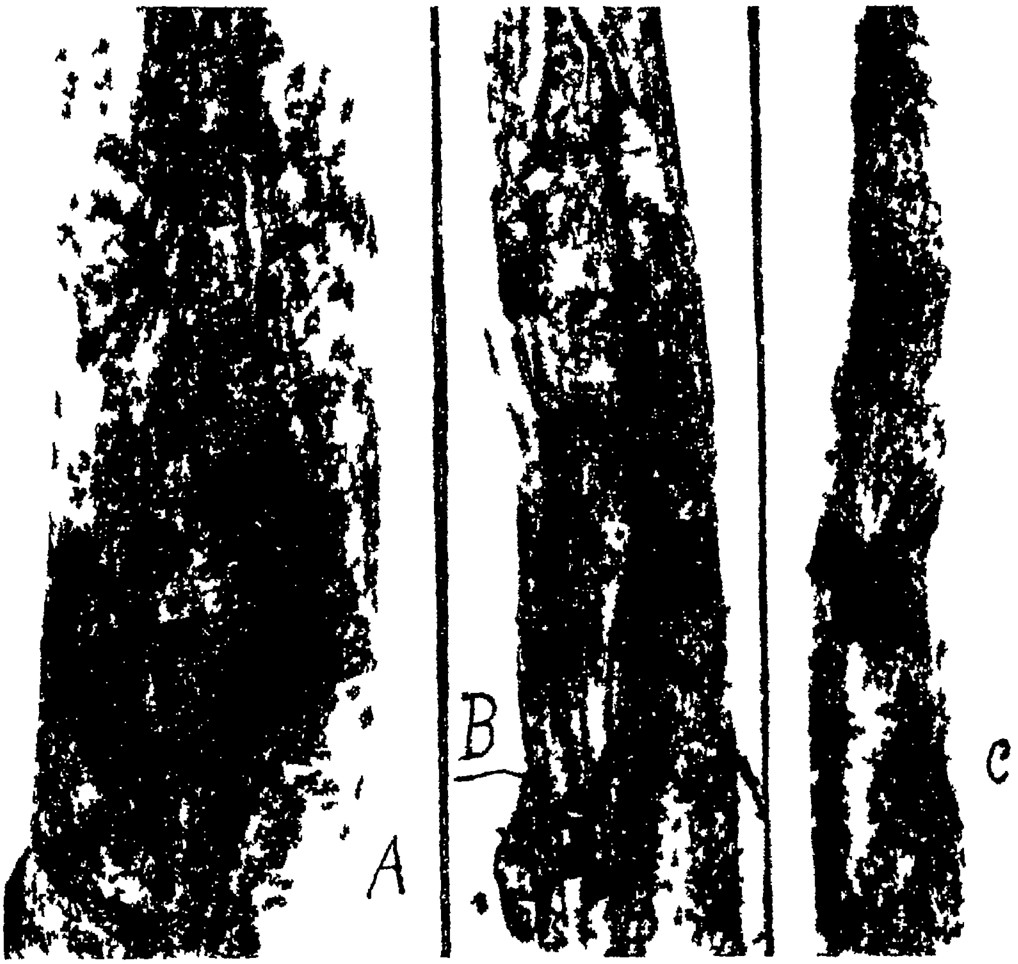

Fig. 11. An Enlarged View of the Cankers Shown in Figure 10

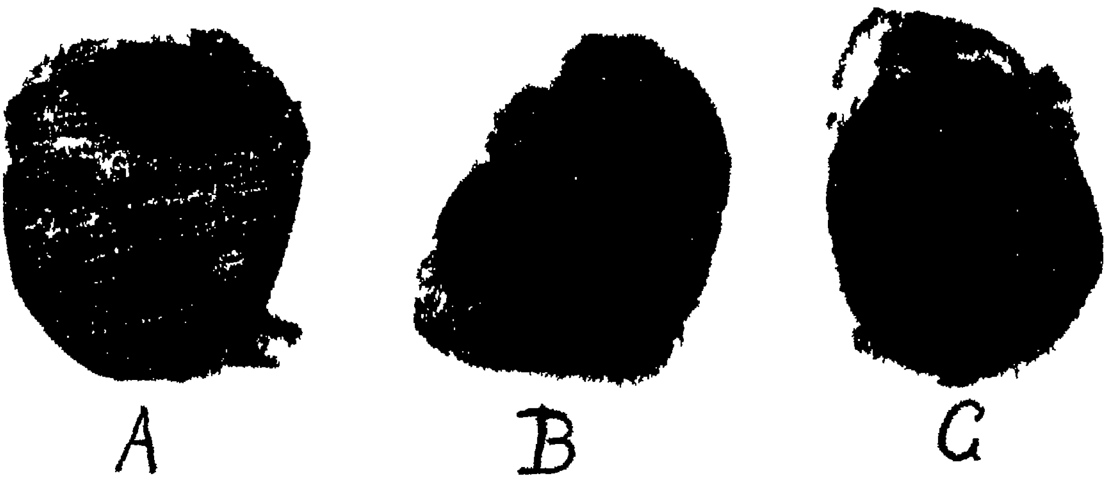

Fig. 12. Cross Section through the Cankers Shown in Figures 10 and 11

and imperfect aeration did not materially influence the injury, which had usually begun in a few days and was complete within two or three weeks from the time of treatment."

When unwounded roots were treated with arsenate of lead, little or no result was observed. When applied to wounds produced by breaking off small rootlets, the injury was usually slight but definite, and in a short time, varying from two to five weeks, small brownish patches appeared, which gradually increased in size, but permanent cankers were produced less frequently than when the arsenate was applied to wounds in the bark.

### WATER LENTICELS

Grossenbacher (18), working in New York State, found that trees affected with crown-rot were most prevalent in cultivated orchards and on thin, stony or gravelly soil. Quite the contrary is true in Yakima; although the disease is found in clean cultivated orchards and on deep soil, it is more prevalent on low, rich soil, especially where there is or recently has been a cover crop of alfalfa kept well irrigated.

It should be remembered that most of the experiments recorded in this article were carried on in orchards with a cover crop of alfalfa. In irrigating, the orchardist often thought of the immediate needs of the alfalfa rather than the trees. Not infrequently there were found at the crown of trees small whitish or slightly yellowish patches which were soft and spongy and which proved to be water lenticels such as are commonly seen when a willow wand is left submerged in water. When the arsenate was applied to these hypertrophied patches marked results were obtained. The large conspicuous cells collapsed, and instead of a swollen area there appeared a circular depression, the center of which eventually became a brownish powder. These affected areas in time often ran together, forming one large discolored space, which in some cases penetrated to the cambium. When these were kept moist cankers were formed. In some instances, however, after the alfalfa had been cut and the region about the treated area had become somewhat dry and aerated, the wounds, even if they had reached the cambium, dried up, leaving only small sunken areas in the bark, which did not seem to be detrimental to the tree.

It was found on further search that these excrescences were quite prevalent in orchards having a cover crop of alfalfa, which, when well irrigated, provided an almost saturated atmosphere at the base of the trees. This humidity, associated with the extreme heat of the Yakima Valley, made an excellent environment for the development of this condition, which was observed most abundantly during July and August, especially on trees that had been severely pruned.

In this search trees were not infrequently found having the bark at the crown, usually just below the ground, soft like rubber and scattered over it a number of more or less circular brown dead patches in which the bark had largely disintegrated into a brownish powder. This is almost precisely what was obtained when water lenticels were treated with arsenate of lead. A photograph of a tree seven or eight years old showing this condition is shown in figure 13, where the brown powdery material has been removed to show the depth of the depression, which in the larger ones extends to the cambium.

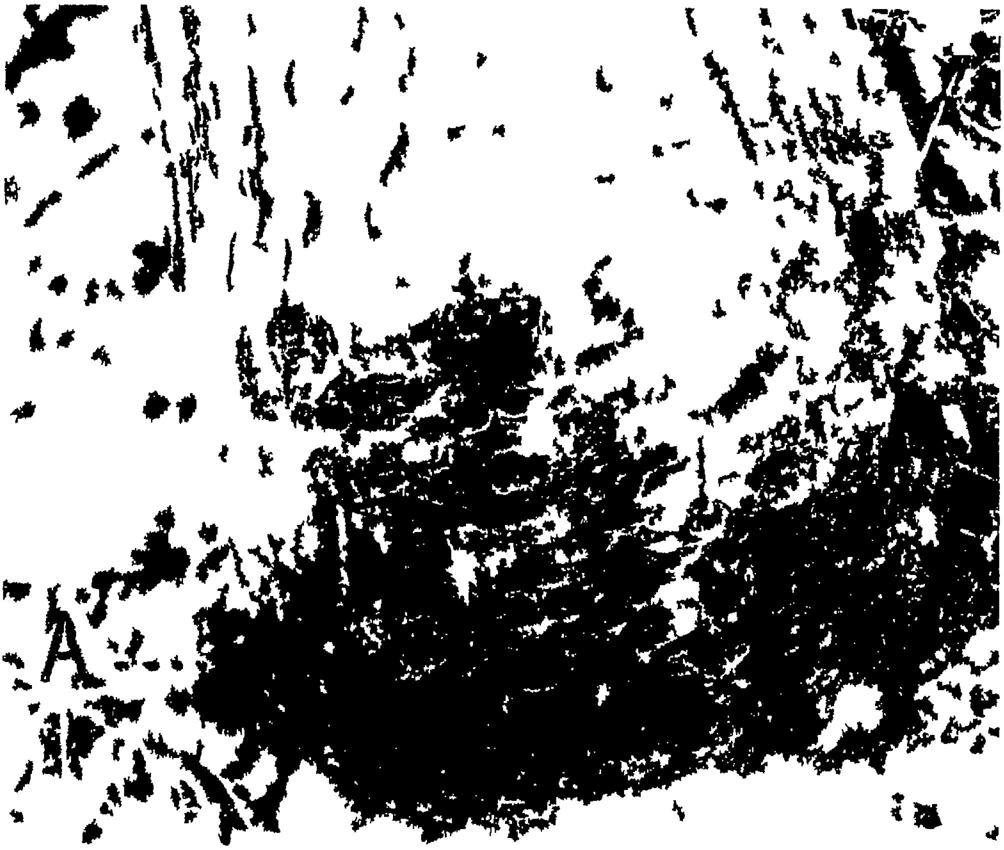

Fig. 1. Early Stages of Arsenical Poisoning

The lower portion of this figure at (a) shows these discolored areas run together, the outer portion of the bark becoming disintegrated, producing a brownish powder when rubbed between the thumb and finger. Beneath this is sound bark except in certain spots which have reached the cambium and discolored it slightly. On further search an almost perfect series has been observed beginning with the isolated brown spots which enlarge, run together and form larger brown patches but still quite shallow, while more advanced stages penetrate entirely through the bark, discoloring the wood just beneath.

These observations show that while the early stages of the lesions caused by frost and winter injury, as shown by Grossenbacher (22), begin at the cambium or inner phloem and eventually cause the bark, which subsequently dies, to be separated from the wood, the lesions caused by arsenate of lead have their initial stages at the outer bark, either at some wound or at such hypertrophies as water lenticels. The corrosion thus begun at the surface of these excrescences gradually destroys the cells and eventually reaches and kills the cambium, producing a permanent canker.

The results of the present investigation as well as those recorded by Headden (23, 24) and Swingle and Morris (37), show conclusively that arsenate of lead will produce definite injuries to the bark of fruit trees. There is little doubt also that a typical collar-rot condition can be produced by the application of commercial arsenate of lead to fresh wounds or water lenticels found at or near the crown. Because cankers are produced artificially when a considerable amount of arsenate is used is not, alone, an absolute proof that it does so in nature, when there is a much smaller amount present. Yet, when a series of conditions practically identical with the artificial injuries are found in nature, it seems highly probable that the arsenate of lead such as is commonly used by orchardists is capable of becoming the primary cause of these cankers.

The writer has produced similar cankers on fruit trees by using copper sulphate, creosote, turpentine, etc. Even with such weak toxic substances as boric acid, potassium chloride, borax, and even sodium chloride, cankers can be made by using concentrated solutions on fresh wounds. This would indicate that the presence of alkali in the soil may perhaps greatly augment the work of the arsenate, not only in increasing the solubility of the arsenate, as shown by Headden (23), but even by its chemical effect on the living tissues of the tree. Considerable alkali, which in the Yakima Valley always contains bicarbonate and carbonate of sodium and frequently the sulphate and the chloride, is always present in this soil. A concentrated solution of these compounds was made and applied to fresh wounds of apple trees in Seattle, producing definite and serious injuries to the trees. It would thus appear that although arsenate of lead is capable of producing permanent cankers itself, yet there is often a combination of chemical factors at work producing this result, and of these the most important one is probably the arsenic.

Moreover, observational evidence leads the writer to the belief that moist soil, especially if alkali is present, when kept more or less constantly in contact with a wound, contributes more toward producing a permanent canker than has been generally accepted. The fact that a wound, from whatever cause, fails to heal indicates that the cambium over the surface is dead. Water containing a relatively high percentage of alkali and probably other minerals from the soil may then gradually soak through the dead tissue into the heart of the tree, following along the dead medullary rays, discoloring the cell walls and eventually producing a collar-rot canker.

### WOOLY APHIS

The development of the primary lesions into typical collar-rot conditions depends largely on the persistence of the factors which prevent the healing of the wounds. It would appear from the present observations that one of the chief agents at work hindering these wounds from healing is the woolly aphis. These insects were found in connection with nearly all forms of collar-rot, but especially in the earlier stages of its development. They readily attack apple trees of all ages and varieties but in general the young trees suffer most. As is well known, the favorite location for their operations is at the crown of the tree just where collar-rot develops. In 75 per cent of the young stages of collar-rot examined by the writer, woolly aphids were found in large numbers. In these cases it would appear that the injury having been made, the aphids occupy the wound, persistently attacking the fresh margin and thus apparently becoming an important factor in preventing it from healing. To what extent these aphids are capable of producing the original wound was not determined, but it is quite evident that when once an abrasion is made their activities tend to prevent the wound from healing, thus contributing towards a permanent canker.

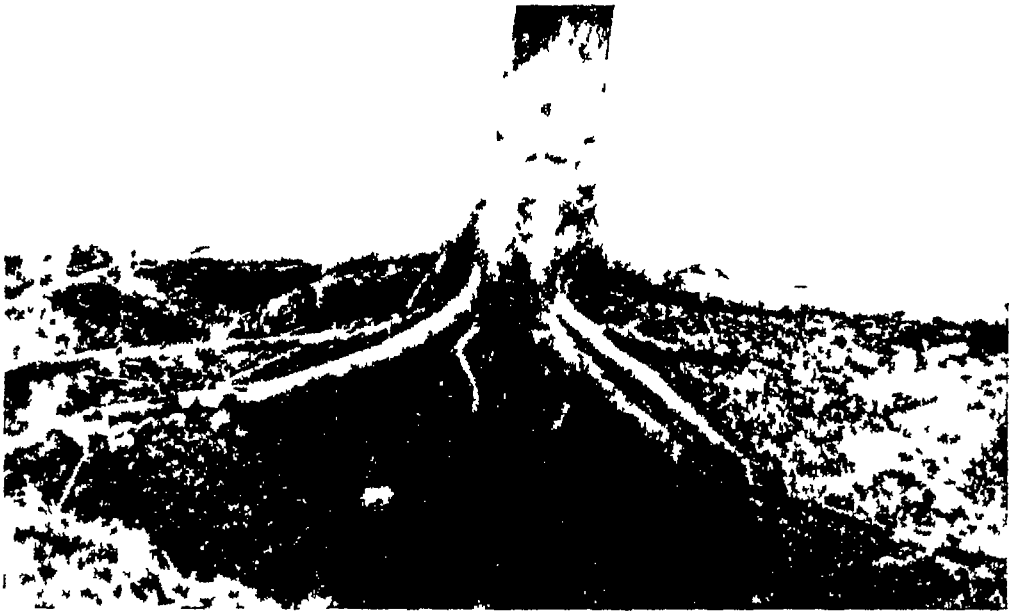

FIG. 14. A TREE COMPLETELY GIRDLED BY COLLAR-ROT

## TREATMENT

In attempting to treat collar-rot the soil was removed, laying bare all the affected parts. These were cleared of all dead bark and thoroughly washed with water in order to obtain a clean surface and then disinfected. A weak solution of lysol, about 1 per cent, was found the most effective, although corrosive sublimate, weak copper sulphate, Bordeaux mixture, lime-sulphur, and formaldehyde were also used. The superiority of the lysol seems to be due to the fact that it penetrates the tissue of the tree a certain distance without any apparent injury. Its effect is thus more permanent. After the wounds were all treated they were left exposed to the air for two or three months. Before the rains came in the autumn the excavations were filled with sand, gravel or cinders, thus facilitating drainage.

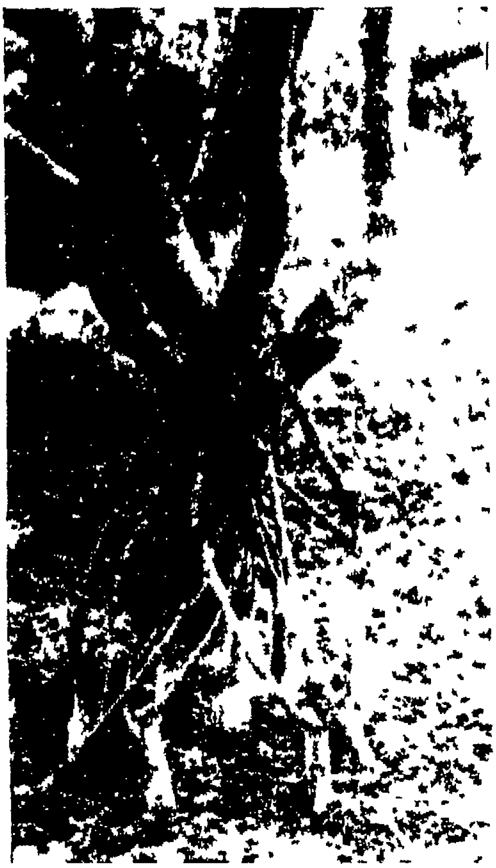

FIG. 15 AN ATTEMPT TO SAVE THE TREE SHOWN IN FIGURE 14, BY PLANTING SEEDLINGS AROUND IT AND GRAFTING THE ENDS OF THE BRANCHES INTO THE SOUND BARK ABOVE

It would be wise for an orchardist to go over his whole orchard once in two or three years with a trowel or shovel, remove the soil from the crowns, and determine if the collar-rot had started. If there is any indication of the disease, the dead bark should be removed and the wound disinfected and finally left exposed to the air for several months.

Bridge grafting has been indulged in at times in an attempt to save valuable trees. The number of scions used varied with the size of the tree, frequently too few being used. The unions were made just below the bark in the sound tissue of the stem above and of the root below the canker. Occasionally when too much of the root was destroyed, young seedlings were planted around the tree, as shown in figure 15, and the ends of the branches cut off and grafted into the sound cambium above. The same tree just before the operation is shown in figure 14. It can be seen that the greater part of the root system is dead, hence the necessity for using young seedlings. Bridge grafting as a method of treatment for collar-rot can hardly be said to be a success. Although a fair percentage of the trees live, they are seldom thrifty, and both the quality and quantity of the fruit they produce are inferior. Moreover, since a large space of the dead stem of the tree is usually left exposed, the tree eventually contracts heart-rot, which forecasts its early death. In the majority of cases, therefore, it would seem more profitable in the long run, where the injury is severe, to remove the affected tree and after a year or two to plant another.

## SUMMARY

1. It would appear that the only condition essential for the development of the so-called collar-rot or crown-rot is a permanent wound of the bark at or near the crown of a fruit tree. As far as the results are concerned it matters little as to the cause of these lesions.
2. A number of different factors may be involved, either as primary causes or contributing causes, in the development of the collar-rot condition.
3. Most of the numerous attempts to isolate definite organisms from the collar cankers have resulted negatively.
4. In a few cases such organisms as *Armillaria mellea*, *Polystictus versicolor*, and *Bacillus amylovorus* have been definitely isolated.
5. The great majority of cankers whose primary cause could not be directly determined were probably initiated by the fire-blight organism, a conclusion arrived at by a comparison of such cankers with those known to have been begun by *B. amylovorus*.
6. Other causes, such as gopher injury, cultivation injury, frost or winter injury, defective graft unions, hard-pan root injury, and arsenate of lead injury, may be responsible for the initial development of collar lesions.[3]
7. The development of the primary or original lesion into a typical collar-rot condition depends upon the persistence of factors which prevent the healing of the wound.

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[3] Since this article was written further work on this disease has brought out evidence that the gradual corrosion of the bark under certain conditions might be the result of a more or less continuous oxidation, the oxidases acting either as the primary causes or contributing factors. This hypothesis is based on the following observations

1. There is an oxidase present in the bark as shown by the bluish color produced by guaiacum.
2. The diseased bark is less acid than the healthy bark.
3. Oxidases tend to work more rapidly where the acidity is weak. The presence of constant moisture more or less charged with alkali reduces the acidity in the immediate vicinity of the abrasion, thus making the conditions more favorable for oxidation.

8. Observational evidence leads the writer to the belief that there are at least two important factors at work in preventing the normal healing of the crown-lesions and therefore in promoting the characteristic disintegration of the tissue known as collar-rot. These are the presence of the woolly aphids and a saturated condition of the soil in constant contact with the primary lesions, particularly if the soil water is charged with alkali salts.

9. The only method of treatment thus far devised is the cutting out of diseased tissue, the washing of the wound, disinfecting it, and finally leaving it exposed to the air. Where injury is severe when discovered, removal of the tree is probably wise.

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# A PRELIMINARY STUDY OF THE PURPLE LEAF SHEATH SPOT OF CORN

L. W. DURRELL

WITH SIX FIGURES IN THE TEXT

The purple sheath spot of corn is a conspicuous and universally present spot disease, yet its cause has never been definitely determined. It is the purpose of this paper to present observations and experiments demonstrating the cause of this disease.[1]

The prevalence and distribution of the disease is suggested by Burrill's (2) statement, which reads as follows: "From observations now made it appears that the disease is a very prevalent one and probably has existed during the time that corn has been grown on the continent." It has been observed in Iowa by the author and members of the station staff over a period of four years. During the latter part of the summer of 1918, counts on 9620 stalks in representative fields showed 86 per cent badly spotted with the purple leaf sheath spot.

The characteristic symptom is a purple or brown spotting or blotching of the leaf sheaths and husks (fig. 1). This condition develops rapidly on all plants after flowering. Examination of the blotches reveals a darkened, disintegrated condition of the tissues on the inside of the sheath. In the center of the larger blotches one usually finds old pollen sacs that have been dropped from the flowers above. In the center of the smaller spots one finds pollen grains that have been washed or shaken down inside of the sheath, as shown in figure 2.

The purple sheath spot of corn was first definitely described in 1889 by Burrill (2 and 3), who attributed it to a bacterial organism. He writes of it as follows: "The first indication of the disease in a field . . . . is the dwarfed condition of the young plants. . . . . It is found in the rich spots rather than in those of poorer quality. In many cases it is upon the lowest ground. . . . . Upon closer observation it is found that young diseased plants, besides being smaller . . . . are uniformly yellowish in color. . . . . The bottom portion of the stalk is likewise affected. . . . . If split longitudinally . . . .

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[1] The author wishes to acknowledge the many helpful suggestions made during the progress of this study and the preparations of the manuscript by Dr. I. E. Melhus.

the inner tissue of this lower part is seen to have a uniform dark color. . . . . After midsummer, especially, the disease becomes apparent through discolorations of . . . . the sheaths, which closely invest the stalk. These leaf sheaths become variously spotted as observed from the exterior. Sometimes the watery-brown portions are mere specks; sometimes large irregular patches. . . . . Occasionally there is a little reddish color. . . . . If now these affected leaves are stripped from the stalk, it will be found that evidence of the injury is more conspicuous on their innerside. The injured patches . . . . have a more watery appearance and sometimes are more or less smeared with some gelatinous substances. . . . ."

In 1888 Kellerman and Swingle (7) discovered a bacterial spot of sorghum, and sent specimens to Burrill for study. This apparently led Burrill to the investigation of similar spots on corn sheaths, with the subsequent publication of the above description. At this time, too, interest was aroused over a disease of cattle, apparently caused by eating diseased corn stalks. The sheath spot of corn was one of the diseases thought to be responsible; experiments, however, did not confirm this assumption. Billings (1) of Nebraska, in correspondence with Burrill on this subject, writes as follows: "The malady as affecting this staple crop has not been fully worked out because for some unknown reason the attempts to communicate the disease to healthy plants by application of culture materials containing the living organism obtained from diseased plants have not been successful." He further states that no results were obtained by inoculating plants with an organism that he had isolated and considered specific for the cattle disease. Not until August, 1889, did Burrill obtain

FIG. 1 PURPLE LEAF SHEATH SPOT OF CORN

satisfactory results from inoculations with the organism he had isolated from the corn spots, and his success even then was slight, except where punctures were made in the surface of the leaf sheaths.

In the light of our present knowledge of corn diseases, the description he furnishes throws some doubt upon the work, for it would appear that the symptoms as described have been associated also with other corn diseases, such as corn wilt. Writing of corn wilt, Smith (10) says: "The

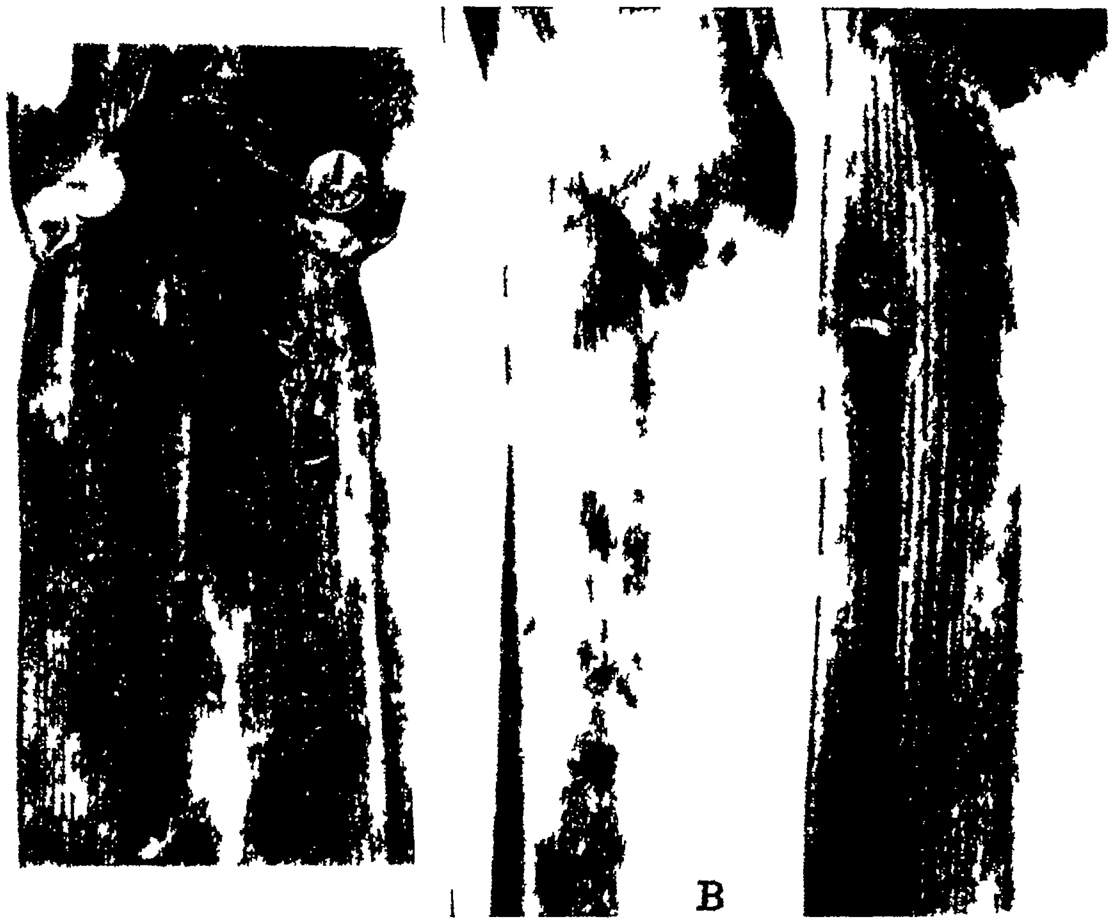

FIG. 7. (*A*) Inner surface of corn sheath showing adhering pollen sacs that have dropped past loosened ligule, and the resulting discolored areas of infection. (*B*) Pollen sacs on inside of corn sheath, and the surrounding fungus-invaded areas.

history of this disease begins with Stewart's bulletin (1897), unless we are to assume that Burrill's bulletin on 'A Bacterial Disease of Corn' (1889) relates in part to the same subject. Some parts of his description correspond very well to Stewart's disease, e.g., irregular distribution of the disease in the field, dwarfing, brown nodes, the base of the stalk most badly affected, death of the leaves as a whole; but if he really had this disease under observation why is there no mention of the most conspicuous sign, i.e., yellow slime in the internodal bundles? . . . . Along with these signs, as a part of the same disease, are mentioned other less characteristic signs, i.e., decaying roots . . . . .; brown spots on the leaf sheaths, most conspicuous within, and occasionally bordered by red. These brown spots are 'half rotten' discolorations, the disease in this stage being evenly distributed through this field. . . . . The description of the organism which he isolated and with which he obtained infections is what finally decided me to exclude it. This was a white peritrichiate schizomycete subsequently identified by Dr. Theobald Smith as *Bacillus cloacae.*"

The dwarfing and decaying of roots of the corn plants described by Burrill suggests the possibility that he had under observation the disease discussed by Hoffer and Holbert (6) in their study of root and ear rots.

Further mention of the sheath spot is made by Rosen (8) in connection with his studies of a bacterial rot of corn. He writes. "The only other diseased condition noted was a dark brown spotting of husks, leaf blades and sheaths. It is possible that this represents a type of secondary injury due to the pathogen responsible for the root rot. It may be mentioned that aphids have been found associated with some of these spots, which may act as carriers of the disease producing organism, also that small, purplish-brown spots resembling the spots found on plants growing in the field have been produced by artificial inoculation with the organism causing root rot."

It is obvious from the above brief review that the cause of the purple leaf sheath spot was not definitely demonstrated by Burrill and has not received serious consideration by subsequent workers.

The author (4) presented an abstract at the St. Louis meeting of the American Phytopathological Society, briefly describing the cause of this disease on the leaf sheaths. In this paper it is planned to present a more complete discussion as to the nature of the purple leaf spot of corn.

## TIME AND APPEARANCE OF SHEATH SPOT AS RELATED TO DISTRIBUTION OF POLLEN

The disease appears late in the season at the time of, or shortly after, blossoming of corn. In the plots of known ancestry in the experiment station fields, where counts were made, practically all of the stalks exhibited spotted sheaths after flowering, and the development of the disease advanced rapidly in all parts of the field after the dropping of the pollen.

This observed fact suggested a possible association of pollen with the disease. Examination of the tissues of the sheath next to the stalk, as previously mentioned, showed a rotted condition, and in every case either pollen sacs or pollen grains were found in the center of the spots (fig. 3).

The leaf sheath of corn completely encloses the stalk with over-lapping edges, and when young the ligule fits tightly. As the plant matures, however, this grasp is relaxed, and the action of the wind on the leaves loosens the ligule and affords a means of entrance for spores, dust, pollen, and organisms of various kinds. Pollen and pollen sacs, falling from the tassel above, light on the leaves in great masses; they are readily washed past the loosened ligule and lodge within the sheath. Centrifuged washings from many leaves reveal numerous spores of different fungi, which, together with the pollen, lodge within the moist enclosure of the leaf sheath. Earlier in the season, the rapid growing stalk forces out anything introduced within the sheath. Bits of fungi on media introduced within the sheath in the evening have been found above the ligule the next day. After flowering, however, there is practically no elongation of the stalk; consequently, pollen and spores remain within the moist enclosure of the sheath, where excellent conditions are furnished for organisms to begin growth on the food stored in the pollen. When this is exhausted they attack the tissues of the sheath. The effect on the host tissue is a corrosion and discoloration of the tissues of the sheath surrounding the stalk. The stalk and the outer part of the leaf sheath are scarcely ever attacked. This is readily explained by the presence of stomata on the inner surface of the sheath, affording an entrance for the organisms, together with the difference in thickness of the cell walls of relative parts, as shown in figure 4.

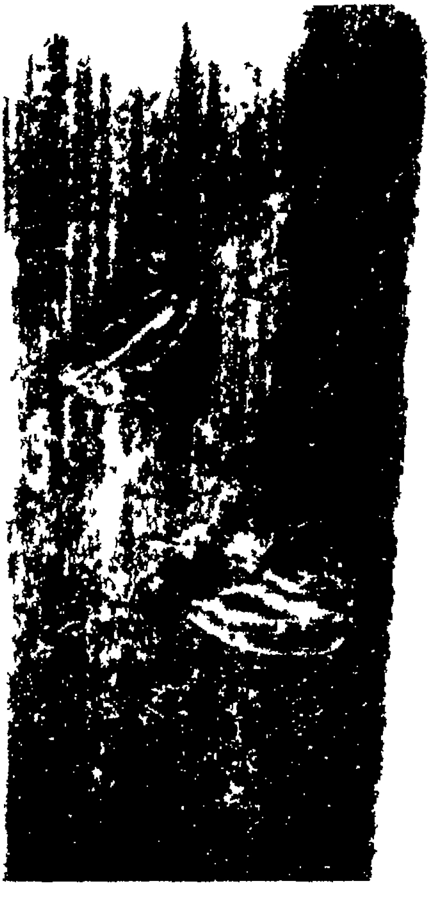

FIG. 3. ENLARGED VIEW OF POLLEN SACS AND RADIATING AREAS OF INFECTION

Produced in greenhouse

INFECTION EXPERIMENTS

Acting on the observational evidence that introduced pollen grains or pollen sacs aid infection, experiments were conducted in the field to exclude these. Cotton gauze was wrapped around the stems of a number

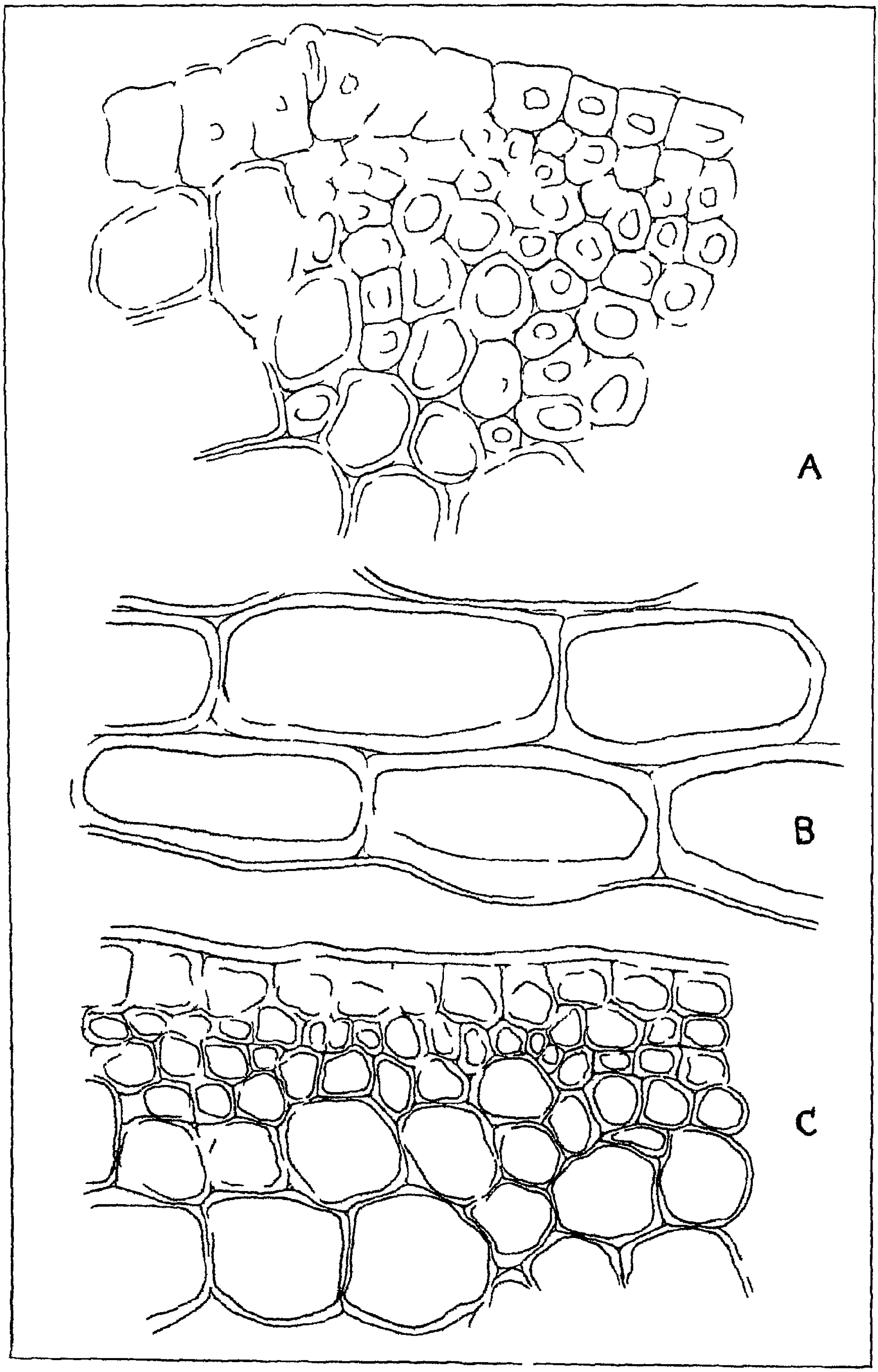

FIG. 4 CAMERA LUCIDA DRAWINGS OF SECTIONS OF SHEATH AND STALK OF CORN

(*A*) Hard outer surface of sheath (*B*) thinner walled inner surface of sheath (*C*) hard surface of stalk adjacent to sheath of plants just above the ligule and tied tightly so as to exclude any pollen falling from above. Where it was tied tightly enough, no spot resulted on the sheaths. Other stalks were detasseled and covered with tall cloth houses to exclude pollen. Under this condition no spotting of the sheaths resulted, while all the neighboring stalks were infected.

In the green house results were comparable to those noted in the field Here experiments were made excluding pollen from the leaf sheaths by. growing the plants away from any source of pollen. Plants thus grown developed no spots on the sheaths. Further, pollen and pollen sacs, direct from the tassel, were introduced within sheaths of corn grown away from any source of pollen and free from sheath spot. Following such introductions the sheath spot soon developed, species of Penicillium usually being the causal agent.

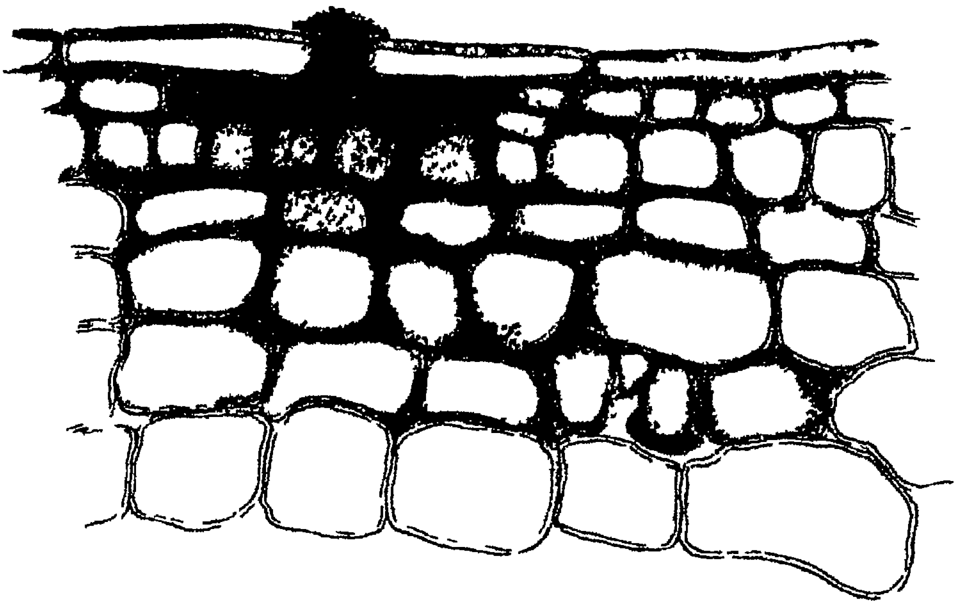

FIG. 5. SECTION OF SMALL DISEASED SPOT ON SHEATH OF SWEET CORN

Showing invasion of bacteria from excretions of aphids and the resulting corrosion of the tissues.

Different organisms were obtained from plantings made from the leaf spot as it developed in the field. From 304 plantings of diseased tissue, species of Penicillium and Fusarium, and several bacteria were obtained. The Fusarium was the most prevalent. This suggested that perhaps more than one organism attacked the sheaths. Based on this assumption, pollen sacs were dusted with spores of the above mentioned fungi, which had been isolated in April, 1918, and placed inside the leaf sheaths of corn growing in the greenhouse. It was found possible to produce the sheath spot with all of the above named organisms. However, it was felt that these experiments were not sufficiently convincing and accurate, due to the unknown agents used as inoculum. In April and May of 1919, corn growing in the greenhouse was inoculated with bacteria and fungi of known species.

Two species of bacteria were used, *Bacillus subtilis* and *B. coli,* and four species of fungi, *Fusarium culmorum, Rhizopus nigricans, Penicillium glaucum,* and *Helminthosporium sativum.* All of these organisms in many trials produced necrotic spots on the inner surface of the leaf sheaths if they were applied growing on small bits of media or if their spores were dusted on pollen sacs. The necrotic spots on the inner surface of the sheaths caused the purple sheath spot so prevalent in the field.

These infection experiments of two seasons seem to indicate that many organisms will corrode and discolor the leaf sheaths of corn if given desirable media, such as pollen, to start growth on. Sections of the spots produced artificially by these various fungi show plainly the invasion of the organisms (figs. 5 and 6). When spores or masses of mycelium were placed under the leaf sheaths without pollen or pollen sacs, no spots resulted. If, however small bits of agar were used instead of pollen, purple spots often resulted. Since the organisms causing the spots are mainly saprophytes, it is apparently necessary for them to be given a start on stored food, such as pollen or agar. The organisms seem to kill the cells of the corn plant sufficiently to allow subsequent invasion.

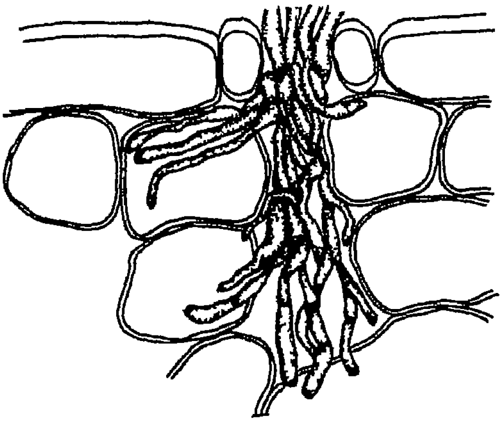

FIG. 6. MYCELIUM OF MACROSPORIUM SP. ENTERING STOMA OF CORN SHEATH

It has been noted further that the sugary excretions of aphids act in the same capacity as pollen; organisms grow on it and later penetrate the corn tissues. Many instances of distribution of organisms by insects are extant, and the carrying of spores by ants is well known. Forbes (5) thought the chinch bug in some way responsible for the corn sheath spot. In the case under consideration there seems to be little doubt that ants aid in the production of the sheath spot. It was frequently observed that the ants placed the aphids within the corn sheath even when it seemed quite tight. In this way they act as carriers of bacteria or spores. The punctures of the aphids afford added means of entrance for the organisms.

## SUMMARY

Corn is universally affected by a disease producing purple blotches and spots on the leaf sheaths.

These spots are an external manifestation of necrosis of the tissues beginning on the inside of the sheaths, appearing only after the pollen has fallen. On the dead spots are pollen and pollen sacs, which seem to serve as media for various bacteria and fungi. These organisms seem to live saprophytically on the pollen at first, and later attack the adjacent host cells of the corn.

Sections of purple spots show stomatal invasion of bacteria and fungi and subsequent necrosis of the tissues.

The introduction of pollen or pollen sacs dusted with spores of molds and bacteria within the leaf sheaths produces the spots.

Spores of fungi alone when introduced within the sheaths do not produce spots, but if bits of agar are introduced spotting occurs.

Exclusion of pollen from the leaf sheaths prevents the development of the purple leaf sheath spot.

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# PHYTOPATHOLOGICAL NOTES

*The summer field meeting of the American Phytopathological Society.*—The American Phytopathological Society has successfully continued the custom inaugurated last year of holding a summer field meeting under the management of the Advisory Board of American Plant Pathologists. Last year's meeting was held at Riverhead, Long Island, June 24–27, 1919, with special reference to potato diseases. The meeting this year, which was especially concerned with apple and peach diseases, was held August 3–6, beginning at Staunton, Va., and extending northward through West Virginia and Maryland to Gettysburg, Pa., one day being spent in each of the four states.

Local arrangements were in charge of the plant pathologists of the states visited, in coöperation with the county agents, the local associations of fruit growers and other local organizations. These local committees were markedly successful in anticipating all needs and in providing all necessary facilities for the transportation and entertainment of the large party of visitors. Many demonstrations and exhibits of fruit diseases were prepared as well as special comparative spraying and dusting experiments on the control of various diseases. Transportation from point to point was by automobiles provided by the growers' associations, with occasional short journeys by rail.

August 3 was devoted to a tour of the Staunton-Harrisonburg district in Virginia, and included the inspection of interesting demonstrations of apple root rot and cedar rust, and comparative dusting and spraying experiments for control of various apple diseases. The party spent August 4 in Berkeley County, West Virginia, noting the effects of cedar eradication, visiting orchards where dusting and spraying experiments were in progress, and inspecting demonstrations of collar blight and other diseases. Visits were also made to the experimental packing plant at Inwood, and to the West Virginia pathological weather instrument station near Martinsburg. August 5 was spent in the vicinity of Hagerstown, Md., and was largely devoted to peach diseases and their control by dusting and spraying, some attention also being given to truck crop diseases. On August 6 the party visited the Field Laboratory of the Pennsylvania Agricultural Experiment Station at Arendtsville, Pa., and inspected experiments in progress in that region under direction of the laboratory staff on the control of apple diseases and insects. The conference adjourned at Gettysburg but on

August 7 a portion of the party continued by automobile to Philadelphia visiting the rich agricultural districts of Lancaster County and inspecting the tobacco experiments in progress there.

About seventy-five plant pathologists, including a number of horticulturists, entomologists and other specialists representing the United States Department of Agriculture, and the experiment stations of the United States and Canada attended the conference. In addition, the Federal Government detailed specialists from the Geological Survey, the Bureau of Soils and the Bureau of Chemistry to attend the conference and participate in the discussions on the relation of soils to plant diseases. The evenings were devoted to informal meetings, where the growers joined the visiting scientists in reviewing the day's tour and in discussing fruit disease problems. An unusual opportunity was thus afforded all those in attendance, not only to observe the fine orchards and to gain a knowledge of the apple and peach industry in one of the most famous fruit growing districts of the eastern United States, but also to observe many important diseases of those crops, to study their control, and to discuss the problems involved with specialists in their own and in related fields.

A noteworthy feature of these pathological field meetings is the presence of foreign plant pathologists who are in attendance as the official representatives of their respective governments. Last year three well known potato pathologists attended the field meeting at Riverhead—A. D. Cotton of England, G. H. Pethybridge of Ireland, and H. M. Quanjer of Holland. This year the following official delegates from England, France, and Italy were present:—W. B. Brierly, Head of Department of Mycology, Institute of Plant Pathology, Rothamstead Experiment Station, Harpenden, England; Etienne Foex, Directeur de la Station de Pathologie vegetale, Paris, France; G. Rossati, Royal Agrarian Delegate to the Italian Chamber of Commerce, New York City; and K. Nakata, Professor of Plant Pathology, Agricultural Department, Kiushiu Imperial University, Fukuoka, Japan. The presence of these foreign pathologists not only greatly increases the interest and value of these conferences to their American colleagues, but also lays the foundation for international coöperation of inestimable importance to the progress of plant pathology.

The choice of locality for the 1920 phytopathological field meeting was especially fortunate. Agriculturally, the region visited is one of the most important of the United States. Scenically, the Shenandoah Valley and the Blue Ridge Mountains are famous throughout the world. Historically, every foot of ground traversed is of interest from Staunton on the south to Gettysburg on the north, and the party was able to make special pilgrimages to the battle-fields of Antietam and Gettysburg. And finally, the kindness of the people in the sections visited, their generous hospitality, and their untiring efforts to make the phytopathological field meeting of 1920 both enjoyable and profitable, will remain a pleasant memory with all those who had the good fortune to be present.

G. R. LYMAN,
*Chairman, Advisory Board of*
*American Plant Pathologists*

*Stem-end rot of greenhouse tomatoes.*—Late in January, 1920, the two southern commercial greenhouses at Macdonald College, Quebec, were planted with tomatoes. On May 2 a profuse growth of *Botrytis sp.* was observed on the stem end of a ripe tomato at the northwest end of the northern house. An examination of the surrounding plants revealed the fact that eight plants were affected. All showed grayish, slightly sunken areas on the stem similar to those described by Stone.[1] They were usually about a foot above ground, and varied in size from ½ to 4 inches in length and from ½ inch to a two-thirds girdling of the stem in width. Growth of the fungus on the stem was slow and sporulation weak in the greenhouse, but in a moist-chamber infected stems gave a profuse mycelial growth and sporulation was at a maximum. Cross sections of the diseased stems showed that the tracheae were discolored and in most cases the lumena were partially filled with a yellow-brown secretion. An interesting feature was the fact that the glandular trichomes characteristic of tomato and potato also showed this discoloration, while the epidermis and cortical tissues were apparently still normal. This was so marked that at a casual glance the stem would appear to be covered with minute sclerotia.

In addition to stem infection, by far the most infection occurred on the calyx, fruit stalk, and stem end of the fruit, and in these cases sporulation was much more pronounced in the greenhouse. As the fruit develops the sepals curl back on the pedicel, and an area about 2 mm. wide around the stem end of the fruit is exposed. It is in this area that infection of the fruit mostly occurs. Cook and Schwartze[2] point out that in blossom-end rot of greenhouse tomatoes caused by *Botrytis sp.* the disease was most common on the roughened blossom ends. Once the calyx is infected fruit infection rapidly follows, whether the fruit be green or mature. Sporulation is profuse (fig. 1) and as the mycelium advances it is preceded by a characteristic water-soaked area, which is well shown in figure 1.

The two houses were connected for ventilation purposes, air movement being maintained by the removal of areas of glass in the common wall of the two houses. The door communicating with the corridor was at the end of the greenhouse where infection was first observed, and the opening of this door at any time in the spring gave rise to a strong current of air through the two houses. There was at the same time an infestation of white fly (*Aleyrodes vaporariorum* Westw.). Without doubt the opening of the door caused hundreds of the flies to be carried throughout the houses and spores were distributed by these carriers. By May 15 the disease had spread through both houses, very few plants remaining free from infection.

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[1] Stone, R. E. A new stem-rot and wilt of tomatoes. Phytopathology 9: 296–298. July, 1919.

[2] Cook, M. T., and Schwarze, C. A. Two interesting diseases of greenhouse tomatoes. Phytopathology 6: 364–366. August, 1916.

When the fruit was attacked it would in most cases fall, unless it happened to be in the middle of a bunch, when it would be retained in place by the others. The other fruits in the bunch would become diseased later, in all probability through mycelial infection.

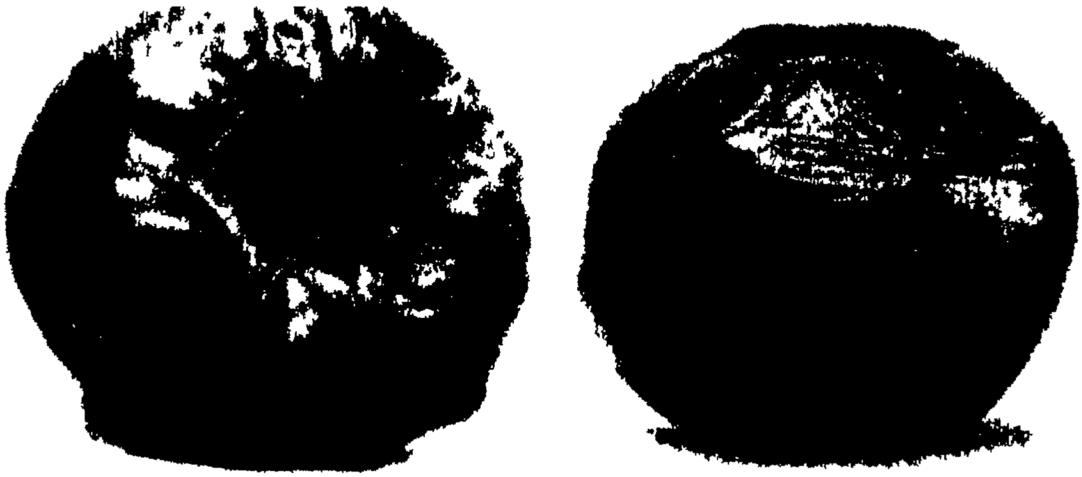

FIG. 1. STEM-END-ROT OF GREENHOUSE TOMATOES

Showing at the left profuse sporulation of Botrytis sp on the stem end of tomato. The surrounding water-soaked area is also shown. The right hand fruit shows clearly the water-soaked area caused by the advancing mycelium. Both natural infections.

Pure cultures of the fungus were made on sterilized young tomato-stem plugs, on green tomato, on ripe tomato, on tomato leaves, and on glucose agar. In all cases there was luxuriant mycelial growth followed by abundant sporulation. At first the mycelium was grayish-white but as the culture aged it became grayish-brown. The spores were egg-shaped, cinereous, and large, being on an average 8$\mu$ by 12$\mu$.

No sclerotia developed on material in the greenhouse, and in laboratory tests they developed in one case only. On May 3 a culture was made on a sterilized young green fruit, and by May 28 there were sixty-three small black sclerotia averaging 1 mm. in diameter.

No wilting of the plants occurred at Macdonald College, as no case was observed of complete girdling of the stem, but the fruit infection assumed serious proportions.

B. T. DICKSON

MACDONALD COLLEGE
QUEBEC, CANADA

*Sclerotinia wilt of greenhouse tomatoes.*—On the morning of April 26, 1920, my attention was called to a badly wilted tomato plant in one of the commercial greenhouses at Macdonald College, Quebec According to the greenhouse attendant, the plant was apparently in good health on the evening of April 24.

On examination the base of the stem was found to have a slight covering of whitish mycelium for a distance of 6.5 cm. above ground. At this height above the soil level there was a slight injury where the first leaf had been removed, probably during cultivation.

Upon opening the stem three very fine black sclerotia were found, together with a little white mycelium, occupying the center in place of the pith. All the pith was gone up to a height of 10 cm. above soil level and, also, part of the woody tissues. The uppermost sclerotium was 7.5 cm. and the lowest 3 cm. above ground level. They measured respectively 3.5 mm. by 14 mm. (fig. 2, *A*), 3 mm. by 7 mm. (fig. 2, *B*), and 4.5 mm. by 8 mm. (fig. 2, *C*).

One sclerotium was washed in $HgCl_2$ solution and then in distilled water and planted in a test-tube containing sterilized green tomato fruit, on May 3. It was kept at room temperature, and by May 15 there was a profuse growth of white mycelium typical of *Sclerotinia libertiana* Fcl. On May 17 sclerotia to the number of sixteen had developed in the culture. They were similar to those which developed in the tomato stem, and were on an average 3.5 mm. by 7 mm. in measurement.

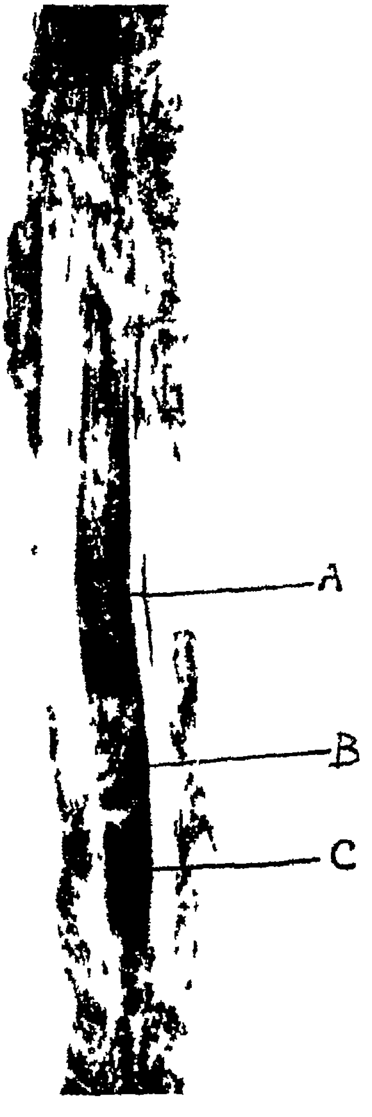

FIG. 2. SCLEROTINIA WILT OF TOMATO

Opened tomato stem showing three sclerotia of *Sclerotinia libertiana*.

By May 8 the plant standing next to the one first discovered succumbed in a similar manner. Examination again revealed black sclerotia to the number of five, occupying the medulla of the diseased plant at a height of 16 cm. above soil level. On sterilized tomato plugs these gave rise to abundant white mycelium of *Sclerotinia libertiana,* and later numerous sclerotia developed.

The bed in which the tomatoes were growing had previously been planted with lettuce, which had suffered from an attack of lettuce "drop," and sclerotia had been collected from this part of the bed in December, 1919, on diseased lettuce leaves.

There was no yellowing of the leaves of the plant prior to the wilting, the wilt setting in quite suddenly.

This disease is apparently similar to that of potato known as "stalk disease," which is second only to late blight in importance in many areas of Ireland, and which is stated to be caused by *Sclerotinia sclerotiorum.*

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*A suspected mosaic disease of sweet clover.* Mosaic is now of such wide distribution and economic importance, that its occurrence on a new host is worthy of note. A mosaic condition of sweet clover plants has, no doubt, attracted the notice of many; yet so far as can be learned, no mention has been made of it in literature dealing with plant diseases. In this note it is proposed simply to record its occurrence and the results of certain preliminary experiments carried out in the laboratory during the summer months.

The disease is without doubt quite common on sweet clover. Dr. Gussow states that throughout eastern Canada he has noticed the disease in a great number of localities. It was first seen by him in 1912 in plants in the vicinity of St. Catherines, Ontario. In the vicinity of Ottawa it is of very common occurrence. In almost every patch where the sweet clover was found growing wild, the disease was present. In most cases approximately half the plants were affected. In the plots of the Division of Agrostology on the Experimental Farm, where the plant is grown for certain tests, practically every plant was found to be diseased. It is interesting to note that these plots are the progeny of a limited number of parent plants. These parent plants are still living, and they also show distinct evidence of mosaic being present. It is not known, however, if the parent plants were affected at the time the seed was borne.

Affected plants show all the distinguishing symptoms that characterize Mosaic disease (fig. 3). The leaves have a mottled appearance and are

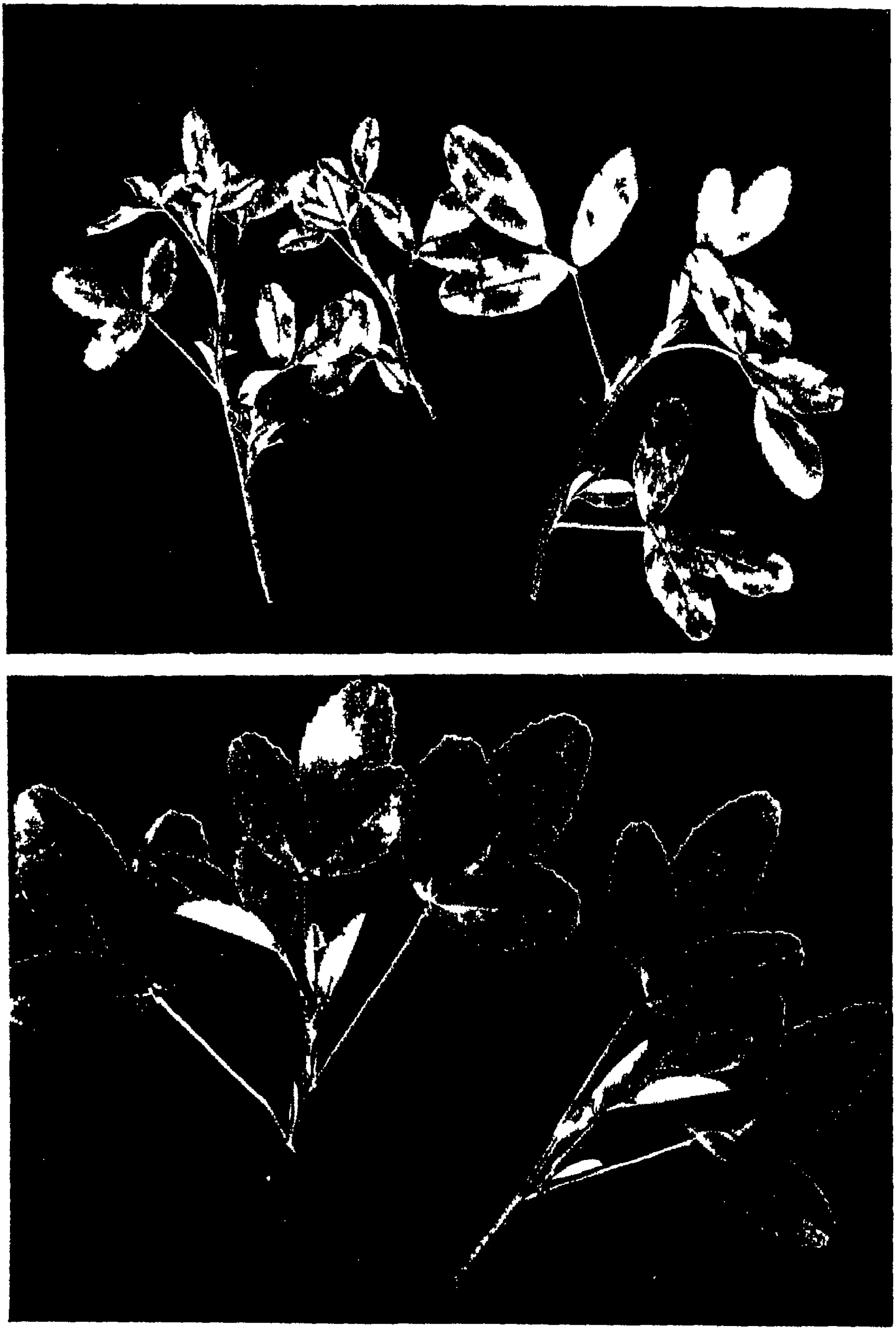

FIG 3 SUSPECTED MOSAIC OF SWEET CLOVER COMPARING PORTIONS OF DISEASED AND SOUND PLANTS (SAME SCALE)

distorted and curled. The plants are somewhat dwarfed and exhibit an unhealthy appearance. When growing wild, diseased plants yield about one-half the herbage of their healthy neighbors. (In the cultivated plots the growth does not appear to be seriously interfered with.) Diseased and healthy plants are commonly found growing side by side. The disease is most apparent in new growth. In severe cases an early defoliation is noticeable.

During the summer months a number of inoculation experiments were made in the greenhouse. Plants for these experiments were grown in pots from ordinary commercial seed. All plants were free from mosaic. Juice was expressed from wild diseased plants and inoculations were made in the leaves by means of a sterile needle. On July 5 a set of twenty plants were thus inoculated; by July 27 twelve were distinctly diseased. On July 6 a set of fifteen plants were inoculated with juice used in former experiments after it had been passed through a Chamberland filter. Four of these plants became infected. Checks to these experiments remained healthy throughout the summer. Other inoculation experiments were made at later dates during summer. Some 140 plants were used in these, but there resulted only three infections. As the same procedure in making the inoculations was followed, the reason for the low percentage of infection is not known.

The writer has had the pleasure of discussing the results of the experiments with Dr. Gussow. He agrees that the disease is in every respect a typical mosaic. The inoculated plants develop the disease in the characteristic manner, i.e., by showing itself first in the new growth, and by not attacking fully formed leaves. He suggests that the particularly low percentage of infection in the later experiments was due probably to a higher temperature at the time of these inoculations, or perhaps to a change in the infectious qualities of the juice as the season advances.

Further experiments are being made, and a report will be made on these as results warrant. The writer wishes to express his appreciation of Dr. Gussow's interest and advice in these experiments.

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*Plant pathology in the Philippines.* The Department of Agriculture and Natural Resources of the Philippine government has recently organized a division of Mycology and Plant Pathology. H. Atherton Lee, formerly pathologist, Office of Fruit Disease Investigations, United States Department of Agriculture has been appointed Mycologist and Pathologist in charge. Mariano G. Medalla, formerly citrus canker agent, Bureau of

Plant Industry, United States Department of Agriculture, has been appointed Assistant Pathologist. Feliciano M. Clara, Felicisimo B. Serrano, and Gaudencio M. Reyes, graduates of the College of Agriculture, University of the Philippines, have been appointed scientific assistants. The laboratories of this newly created division have been established in the Philippine Bureau of Science and very complete laboratory equipment and a botanical library, unusually extensive for the Orient, have been made available for this work through the kindness of Director E. D. Merrill of the Bureau of Science.

*Tropical phytopathology.* The Tropical Research Laboratory of the United Fruit Company, which was formerly located at Zent, Costa Rica, and closed during the period of the war, is being reopened at Changuinola, Panama. Dr. John R. Johnston, Professor of Plant Pathology in the University of Havana, has been appointed Director of Tropical Research for the Company with headquarters in Havana, and two pathologists will be located at the Laboratory in Panama, one to continue work on the banana disease, and one on the diseases of the coconut, cacao and other crops.

*Banana wilt in the Philippines.* Plants of the Latundan variety of the banana, *Musa sapientum*, have been observed affected with banana wilt at Los Baños and Calamba in Laguna Province, and in several municipalities of Batangas Province, Philippine Islands. Affected plants show the typical yellowed leaves, sloughing off of leaf sheaths, split pseudostem, and reddish discolored rhizomes so excellently described by Brandes in Phytopathology, **9**, no. 9, September, 1919. A Fusarium species which agrees very closely with the description of *Fusarium cubense*, E. F. Smith, has been constantly isolated from the upper limits of discolored tissue in the pseudostem. This fungus upon inoculation with needle punctures into the pseudostem of healthy banana plants caused a blackening of the tissues which advanced rapidly longitudinally. Control punctures remained negative. Although the literature concerning the distribution of banana wilt is not all available here, we believe this to be the first observation of this disease in the Philippine Islands.

The disease is found in plantings eight to ten years old and being found in fairly well distributed localities is apparently of long duration in this country.

The abaca plant, *Musa textilis* Née, from which Manila hemp is obtained, is of course very closely related to the banana species. This plant is closely restricted in its distribution to the Phillippine Islands and its products constitute the most important items of export from this country. For this reason we felt considerable concern as to the susceptibility of abaca plants to the banana wilt disease. Inoculations with *Fusarium cubense* upon abaca plants have in our preliminary experiments remained entirely negative, while inoculations from the same culture on banana plants have resulted positively. The evidence indicates that the abaca plant may be considered resistant to this disease.

H. ATHERTON LEE
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BUREAU OF SCIENCE
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# LITERATURE ON PLANT DISEASES[1]

COMPILED BY ALICE C. ATWOOD, AND FLORENCE P. SMITH, BIBLIOGRAPHICAL ASSISTANTS, BUREAU OF PLANT INDUSTRY

## August, 1920

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All authors are urged to cooperate in making the list complete by sending their separates and by making corrections and additions, and especially by calling attention to meritorious articles outside of regular journals. Reprints or correspondence should be addressed to Miss E. R Oberly, Librarian, Bureau of Plant Industry, U. S. Dept Agric., Washington, D. C.

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# STUDIES ON THE RATE OF GROWTH AND BEHAVIOR OF THE BLISTER RUST ON WHITE PINE IN 1918

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The field studies upon which this paper is based were conducted by the writer at Kittery Point, Maine, during the spring and summer of 1918, as part of the campaign of intensive field investigations on the white pine blister rust directed by Dr. Perley Spaulding of the U. S. Bureau of Plant Industry. These studies were undertaken with a view to determining the rate of growth and behavior of the blister rust cankers, and to gain a better conception of the ecology of this very important parasite. Although based on the work of but a single year in a single locality, the results in general are believed to be typical of the rate of growth and behavior of the blister rust on white pine.

## EXPERIMENTAL METHODS

During the last week in April, the beginning of aeciospore dissemination, and the first week in May, one hundred and thirty-three cankers, ranging from the smallest to the largest on the trunks, branches, and twigs of one hundred trees, were outlined by a narrow band (about $\frac{1}{8}$ inch in width) of white lead paint.[1] Each canker was marked by a number painted near it, and the location of each of the one hundred trees bearing the cankers outlined for study was plotted to scale on a map.

The trees in question ranged in age from nine to twenty-five years, in diameter from $\frac{5}{8}$ inch to 9 inches at 1 foot elevation, and in height from $2\frac{1}{2}$ to 30 feet; they were mostly trees in the open that showed little or no tendency to suppression. In addition to the tree description, a full description was made of the individual cankers at the beginning of the study. Along with the latter data a study was made of such points as the origin of the infection; its position on the stem, that is, whether it had occurred at the node or internode, and its location by whorls of branches counted down from the top; the center and extent of the actively fruiting cankers by whorls of branches; the size of the diseased stem in contrast to the size of the normal stem estimated at the same point; the extent of both the past and present aecial areas, together with the abundance of aecia on the 1918 fruiting area; and other points bearing on the increase in size, during the growing season of 1918, of the various zones usually exhibited by the cankers during the different stages of their development. In addition to the points enumerated above, full notes were made from time to time on the death of the trees or branches of the trees, resulting from the normal growth of the fungus, or from attacks by insects or secondary fungi.

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[1] No injurious effect of the white lead paint on the bark was noted, and its superficial application is not believed to have had any effect on the growth of the young mycelium at the outer edges of the cankers, since this occurs mostly in the inner portion of the bark. In many cases aecia burst through the bark directly beneath the narrow band of paint.

The cankers were outlined at the outer limits of the etiolated zone of bark, which roughly marks the tips of the invading mycelium. In the early spring there was a pronounced etiolated zone, usually extending well beyond the pycnial zone of the preceding year, on which latter area the 1918 growth of aecia was starting to develop.

The second series of measurements was made during the last week in July, nearly two months after the beginning of the 1918 pycnial development. At this season of the year there was but very little or no etiolated bark to be seen beyond the pycnial zone, since the new pycnial area had developed over the areas previously etiolated on the cankers. A dotted line was painted about the edge of the etiolated zone, or about the outer edge of the pycnial zone in case the etiolated zone did not extend beyond the pycnial zone. Measurements were made up and down, and to the left and right, from the original painted line in case the canker had not by this time completely encircled the stem on which it was developing. In making the measurements the writer was careful to take them between the centers of the two lines; in this way cumulative errors in the measurements, due to the unavoidable thickness of the line of paint, were avoided. The trees bearing the numbered cankers were under almost daily observation from the latter part of April to the end of July.

During the second week in September the writer returned to Kittery Point in order to again outline the cankers and to complete a third series of measurements on their growth. At this time the majority of the cankers were still producing pycnia abundantly.

At the beginning of the study the problem of securing reliable data for an index of the rate of growth of the blister rust was thought to be a long-term proposition, but the advance of the cankers during the growing season of the host was so decided that the results of the first season's study are deemed worthy of publication.

### BEHAVIOR OF PYCNIA AND AECIA

Pycnia develop on the etiolated zone of bark at the edge of the canker, just beyond the aecial zone, which, if present, is developed earlier in the same year. The production of pycnia began the first week in June and continued steadily until September 10, the date when the last observation was made. It has been the experience of others that the production of pycnia continues until the beginning of winter.

Aecia are produced in the spring with the advent of warm weather and, as a rule, are produced in varying degrees of abundance. They occur chiefly on the pycnial area of the preceding year. On some cankers no aecia may be produced, and on others their development may be so great that they not only fruit all over the pycnial area of the preceding year but extend considerably beyond it. In one case observed, within a little over two weeks after the outer limits of a canker had been outlined on the trunk of a tree 2¾ inches in diameter, the aecial area jumped beyond the line and burst out from under the bark which, previously, had neither produced pycnia nor shown any etiolation, and extended for a distance of 3 inches below the lower limit of the canker as originally outlined, thus bringing it down to the ground line.

Early in the season of aecial fruiting the writer started an experiment to determine just how long the individual aecia disseminate spores. On May 3, three hundred pins, each bearing its number on a piece of paper, were carefully stuck into as many different aecia on a total of twenty-two trees representing an even larger number of cankers. The pins were placed only in those aecia that had either just started or were practically ready to break open. By May 20 the peridia of all but sixty-three of the aecia were well ruptured or lost entirely, together with most of the spore mass. On May 29 only nineteen aecia still retained their peridia, and on June 4 only four of these had preserved their peridia. In the latter case the retention of the peridia was abnormal, the entire aecium being either sealed over with a crust of resin or held in place by tightly appressed bark scales. From the behavior of the three hundred aecia under study it is evident that they attain the climax of their development within less than two weeks after the peridia start to break open, and then begin to decline rapidly. Spore production seems to be mostly over with the maturity of the aecium and the rupture of its peridium. After this stage of development is reached the aeciospores gradually become disseminated. Aeciospore dissemination may be regarded as practically at an end within four weeks after its start, the heaviest part of it being over in two weeks.

After the climax of the aecial development has passed, a few sporadic aecia may later develop out beyond the main fruiting zone. Such pustules, as a rule, are of smaller size than the majority of those first formed.

Quite frequently aecia were observed on old aecial areas that had fruited the preceding year, and, in some few cases, even earlier. Such fruiting seems to be confined to isolated areas where the vitality of the bark was not entirely depleted by the previous production of aecia.

On trees where the infection has been of long standing and its point of origin not too high, the aecia develop down the trunk to the ground line, and often continue their development down onto the roots and even fruit several inches below the surface of the duff and soil.

## RATE OF GROWTH OF THE BLISTER RUST ON WHITE PINE

During the period of four and one-half months for which the growth of the blister rust cankers was computed, a number of potential measurements were lost unavoidably. In some cases entire measurements were lost due to the death of certain trees and parts of trees bearing the measured cankers, as a result of the disease itself, insect attacks, and the breaking of branches. An even larger number of measurements of the downward growth of the cankers was invalidated, since many of the cankers near the base of the trunks grew down to the ground line before the end of the season, so that in such cases the complete record of growth in this direction could not be obtained satisfactorily. Of the measurements on the spread of the cankers around the stems, but few were obtained, since most of the cankers had already encircled the stems at the beginning of the aecial season and the majority of the remainder did so soon after the initial measurements were made. The complete growth records available for the whole season from May 1 to September 10 may be briefly summed up as follows:

One hundred and thirteen measurements on upward growth gave an average figure of 2.44 inches; seventy-seven measurements on downward growth gave an average figure of 2.57 inches. The total vertical growth of the cankers, both up and down, therefore averages 5 inches. Twelve measurements of the circumferential growth of the cankers to the left gave an average figure of 1.17 inches; thirteen measurements of the same type of growth to the right gave an average of 1.33 inches. The lateral or circumferential growth of the cankers, both to the left and right, therefore averages 2.5 inches, or one-half that of the vertical growth. Unfortunately, but few measurements of growth in the lateral direction were to be had, due to the rapidity with which the disease encircled the small stems at the infected point.

In general it may be said that the growth of the blister rust cankers is in direct proportion to the size and vigor of the stems on which they occur. Cankers on the lower and usually smaller branches, either shaded or unshaded, develop very slowly. Shading apparently has little effect on their development.

The rate of growth of individual cankers is quite a different matter from the average rate of growth of a large number. A few cankers that made no growth during the period from May 1 to July 31 advanced considerably after that date. On the other hand, a few that made considerable growth during this period made none during that between August 1 and September 10. Still a few other cankers made no advance during the entire summer, although they produced aecia sparingly in the spring. The greatest upward growth noted between May 1 and September 10 on any one canker was 6 inches; the greatest downward growth noted during this period on any one canker was 8 inches.

A comparison of the growth figures for the first three-month period, from May 1 to July 31, shows that the average vertical growth was approximately the same in both directions and constituted approximately 50 per cent of the total vertical growth of the whole period from May 1 to September 10. The average lateral growth of the first three-month period was approximately the same in both directions, but constituted 70 per cent of the total lateral growth of this whole period. Comparison of the lateral growth of these periods, however, is less certain than that of the vertical growth, as the former is based on vastly fewer figures than the later. In general it may be said that the growth of the blister rust cankers for the one and one-half month period in the latter part of the summer was as great as that of the three-month period in the spring and early part of the summer; in other words, during the latter part of the summer growth was twice as fast as that during the spring and early part of the summer. It is likely that the rate of growth of the blister rust on white pine is influenced chiefly by temperature. That temperature is in all probability the chief climatic influence in determining the rate of growth of the chestnut blight fungus (*Endothia parasitica*), a bark-inhabiting parasite, has been shown by Stevens (5).

The above figures for the growth of the blister rust cankers (5 inches for total vertical growth and 2.5 inches for total lateral growth) from May 1, 1918, to September 10, 1918, do not constitute a reliable index of the total annual growth, since more or less vegetative growth of the blister rust fungus may be expected to occur between early September and the end of April, particularly between early September and the beginning of winter. The advent of winter does not completely inhibit the vegetative growth of many of the wood- and bark-inhabiting fungi, since many of them will grow at temperatures of but a few degrees above the freezing point. For example, Stevens (4) states that field observations and laboratory experiments both show that the chestnut blight fungus (*Endothia parasitica*) will grow whenever the temperature rises above the minimum for growth, which, as Shear and Stevens (3) have determined, is about 8° or 9°C. As Stevens (4) states, this apparently is true regardless of the previous temperature and whether the host is dormant or not.

Mr. G. F. Gravatt, while at Kittery Point early in September, 1919, outlined with paint and remeasured a number of the cankers marked by the writer in May, 1918. In a recent communication he reports that the average growth from May 1, 1918 to September 10, 1918, of the thirty-five cankers which he remeasured was 2.7 inches, whereas the average growth from September 10, 1918 to September 6, 1919, was 4.11 inches, based on the same thirty-five cankers measured at the same points. Most all of these measurements were of growth up the stem. Unless a study of the weather conditions, particularly the temperature, shows a great difference between 1918 and 1919, it must be assumed that the blister rust makes considerable growth between September 10 and May 1. It is to be expected, however, that the majority of this growth will take place between September 10 and the beginning of winter. Judging from the remeasurement figures furnished by Mr. Gravatt, the growth figures from May 1 to September 10 given the writer as an average for cankers of various sizes, represent about two-thirds of the total annual growth of the blister rust cankers.

In table 1 the available figures on the vertical growth of the blister rust cankers from May 1 to September 10 have been correlated with the size of the stems on which they occurred. From this condensed presentation of the figures it is evident that the larger the stems, within the limits given, the greater is the growth of the cankers, in a vertical direction at least.

As only a single tree fell in the "4.1–5.0" diameter class, this individual was omitted from the comparison. This table likewise analyzes the rate of downward growth in comparison to that of the upward growth of the cankers. It shows a tendency, in all but the last diameter class, for the downward growth to exceed the upward growth in cankers falling in the same diameter class.

Many of the largest cankers on the trunks and older branches are formed as a result of two or more branch infections which run back into the main stem either simultaneously or at different times. The result is that a canker of unusual length is ultimately formed. The longest canker noted on a branch was on a twenty-five-year-old tree. In the spring of 1918 this canker was already 42 inches long and had part of the old aecial area eaten off by squirrels; during the summer of 1918 it extended its length nearly 4 inches. The two lateral branches, through which infection entered simultaneously, died during the summer, but the main branch with a canker 46 inches in length was still living at the end of the season. It was only 1⅝ inches in diameter but had enlarged to 2 inches at the center of the canker; the age of the infected portion varied from eight to twelve years.

TABLE 1

*Correlation of the vertical growth of the cankers, from May 1 to September 10, with the size of the stems on which they occurred*

| DIAMETER CLASSES OF STEMS | AVERAGE TOTAL GROWTH UP | NUMBER OF CANKERS BASED ON | AVERAGE TOTAL GROWTH DOWN | NUMBER OF CANKERS BASED ON | TOTAL AVERAGE VERTICAL GROWTH |
|---|---|---|---|---|---|
| *inches* | *inches* | | *inches* | | *inches* |
| 0.1–0.5 | 0.75 | 16 | 1.35 | 16 | 2.10 |
| 0.6–1.0 | 1.57 | 24 | 2.06 | 22 | 3.63 |
| 1.1–2.0 | 2.90 | 31 | 3.09 | 22 | 5.99 |
| 2.1–3.0 | 3.03 | 30 | 3.42 | 12 | 6.45 |
| 3.1–4.0 | 3.55 | 14 | 3.42 | 6 | 6.97 |

The longest canker noted on the trunk was one on a 2-inch tree that had enlarged to 2¼ inches at the center of the infection. In the spring of 1918 the canker had attained a length of 39 inches, apparently all this growth resulting from a single infection, and had grown down to the base of the tree, thereby rendering further growth in this direction difficult to measure. The age of the infected portion of the stem ranged from ten to thirteen years. This tree, which had withstood the attack of the blister rust for so long, finally succumbed early in the summer of 1918.

## PATHOLOGICAL EFFECT OF THE BLISTER RUST ON WHITE PINE

White pine blister rust cankers may result in either a swelling or a constriction of the pine stem; both symptoms may be of equal prominence on the different stems. That there is a regular occurrence of, and a definite relation between, the swelling and constriction of the pine stems does not seem to have been pointed out clearly, the two having been regarded largely as pathological symptoms of more or less haphazard occurrence.

The writer's field studies indicate that infected twigs and the smaller stems up to 2 inches in diameter, as a rule, exhibit swelling which is proportionately greater the smaller the stem, and that infected stems of about 2 inches or more in diameter rarely become swollen but either remain normal in outline or become constricted, depending upon the age of the infection. As a rule infected stems of this size retain their normal diameter for a time and become constricted only after the cankers have developed for a long time. With the continued development of the blister rust cankers on these larger stems the constriction becomes more and more pronounced until the girdling is sufficient to completely retard conduction in the stem and its death ensues. The reason for the difference in pathological symptoms, namely, swelling in comparatively young cankers on the smaller stems, and constriction in the case of the older cankers on larger stems, are readily understood when one makes a stem analysis of the lesions.

According to Colley (2), the mycelium of the white pine blister rust is confined almost entirely to the region of the cortex and phloem cells, although the hyphae do enter the rays and occasionally work their way in between the tracheids so that they have been traced to a depth of three annual rings, counting in from the cambium.

In the early stages of the disease the etiolation of the bark and its subsequent swelling are the first prominent pathological symptoms of stem infection. The swelling is confined to the bark alone, and, according to Colley (2), is due to the fact that the abundant mycelium of the parasite forces apart the phloem and cortex cells of the host, rather than to any increased subdivision of the bark cells.

On larger stems of about 2 inches in diameter, where the cankers develop for a longer period by reason of the fact that the stems are not killed so quickly, the formation of the annual ring becomes retarded and the annual accretion of wood at the central transverse plane of the canker diminishes gradually, often in peripheral extent rather than in thickness. On stems of about 2 inches in diameter the loss of normal accretion is often just about sufficient to compensate for the abnormal thickness of the diseased bark, so that the stem still retains a symmetrical outline; consequently no external indication of the abnormality, other than the diseased appearance of the bark, appears in the stem of the tree at the point where the lesion occurs.

On cankers of long standing, which usually occur on stems of two inches or more in diameter, there is a marked falling off in the normal accretion of the wood occurring simultaneously with the progress of the disease. It often happens that the annual ring fails to develop entirely around the stem at the central point of the lesion in a given year, and each successive ring is less extensive until finally, with the death of the tree or even before, none is formed. Such growth results in trees of very irregular outline in cross section at the center of the lesion.

## STEM ANALYSIS OF WHITE PINES KILLED BY THE BLISTER RUST

Early in July a stem analysis was made on twenty-eight white pines killed by the blister rust. The trees dissected were located on the main infection area used for the study of the rate of growth of the cankers. Of these trees a few, also used for the growth study, had died just prior to the dissection study; most of them, however, had been dead for about a year. The tree description and infection data were made as in the growth study, and these data, together with the stem analysis measurements, are presented in table 2.

The stem analysis measurements were obtained by sawing off the trunk through the center of the canker responsible for the death of each tree. The theoretical age of the stem at this point was obtained by a count of the whorls of branches down from the top. In every case this age count was found to agree with the check derived by a ring count of the normal wood, either above or below the canker, plus the number of nodes (usually only one or two) that it was necessary to go above the canker, or minus the number it was necessary to go below the canker to make a cut suitable for determining the age of the normal wood.

The figures in the column headed "Age of wood actually formed at center of canker" were obtained by counting the total number of growth rings on the transverse section made through the center of the canker. As will be noted, they vary greatly according to the direction of the count, since, as previously stated, in old cankers the annual accretion of wood often fails to develop uniformly around the whole stem, and each succeeding ring becomes less and less extensive until finally, either with or before the death of the tree, none is formed.

The figures in the column headed "Probable age of stem at center of canker at time of infection" could be determined in the field only by the appearance of the annual rings of growth as revealed by the smoothed transverse section of the canker through its central point. The irregularity of the growth rings and their impregnation with resin was found to be a fairly reliable index of the presence of the blister rust after it had attained sufficient development to produce aecia more or less abundantly. There is no reason to believe that the incipient stages of the blister rust have any marked effect on the annual accretion of wood, at least none that would be visible macroscopically. The exact date of stem infection, of course, could be told only by means of a microscopic examination of sections of the wood for the presence of the mycelium. Even such refined procedure would perhaps not accurately establish the exact date of infection, for, though it is likely that the mycelium is to be found in the ray cells of the annual ring laid down the same season that infection took place, it might spread in along the medullary ray cells for a ring or two toward the center of the stem before the death. and consequent infiltration of the wood elements might occur at the point of infection. It is evident that the figures given for the probable age of the different stems at the time infection took place are but a rough index of the time when

TABLE 2

*Summary of a stem analysis of white pines killed by the blister rust*

| TREE DESCRIPTION | | | DIAMETER OF STEM IN INCHES AT CENTER OF CANKER | | SPREAD OF CANKER UP | SPREAD OF CANKER DOWN | AGE OF INFECTED PORTION OF STEM, COUNTED BACK FROM THE TOP | THEORETICAL AGE OF STEM AT CENTER OF CANKER | AGE OF WOOD ACTUALLY FORMED AT CENTER OF CANKER | PROBABLE AGE OF STEM AT CENTER OF CANKER AT TIME OF INFECTION | DIFFERENCE BETWEEN THEORETICAL AND GREATEST ACTUAL AGES OF STEM AT CENTER OF CANKER | YEARS REQUIRED TO KILL TREE AFTER CANKER SPREAD TO MAIN STEM. | AVERAGE DIAMETER OF INFECTED WOOD AT CENTER OF CANKER |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Height | Diameter at height of 1 foot | Age | Estimated normal diameter | Actual diameter | | | | | | | | | |
| *feet* | *inches* | *years* | *inches* | *inches* | *inches* | *inches* | *years* | *years* | *years* | *years* | *years* | *years* | *inches* |
| 10.0 | 3.2 | 16 | 3.5 | 3.2 | 16.0 | 12.0* | 10–16 | 12 | 12 | 7–8 | 0 | 4–5 | 2.7 |
| 10.0 | 2.0 | 14 | 2.7 | 2.0 | 10.0 | 12.0 | 10–13 | 11 | 9 x 10 | 7–8 | 1 | 3–4 | 1.9 |
| 10.0 | 2.0 | 14 | 2.5 | 1.9 | 12.0 | 17.5 | 10–13 | 11 | 8 | 5–6 | 3 | 5–6 | 1.6 |
| 7.5 | 3.0 | 13 | 2.5 | 3.0 | 12.0 | 11.0* | 9–13 | 11 | 10 | 9 | 1 | 2 | 2.2 |
| 14.0 | 3.0 | 14 | 3.0 | 2.6 | 12.0 | 14.0 | 10–14 | 11 | 8 x 10 | 7–8 | 1 | 3–4 | 2.2 |
| 12.0 | 2.1 | 15 | 2.5 | 2.1 | 10.0 | 10.0 | 9–10 | 9 | 6 x 7 | 5–6 | 2 | 3–4 | 1.9 |
| 15.0 | 3.4 | 15 | 3.5 | 2.7 | 7.5 | 8.5 | 11–13 | 11 | 7 x 11 | 7 | 0 | 4 | 2.5 |
| 15.0 | 2.2 | 9 | 2.2 | 2.2 | 19.0 | 10.0* | 12–15 | 12 | 9 x 11 | 8 | 1 | 4 | 1.9 |
| 5.0 | 0.6 | 11 | 0.6 | 0.8 | 4.0 | 5.0 | 8–9 | 8 | 8 | 7 | 0 | 1 | 0.7 |
| 9.5 | 1.4 | 12 | 1.4 | 1.4 | 17.0 | 10.0 | 8–10 | 9 | 9 | 8 | 0 | 1 | 1.3 |
| 7.0 | 2.5 | 15 | 2.5 | 2.4 | 10.0 | 8.0* | 10–15 | 11 | 9 x 11 | 8–9 | 0 | 2–3 | 2.0 |
| 8.0 | 2.5 | 14 | 2.6 | 2.5 | 10.0 | 6.0* | 11–14 | 12 | 10 x 11 | 8 | 1 | 4 | 2.1 |
| 8.5 | 3.5 | 14 | 3.5 | 3.5 | 22.0 | 8.0* | 10–14 | 12 | 9 x 11 | 7–8 | 1 | 4–5 | 2.7 |
| 5.0 | 1.1 | 13 | 1.4 | 1.5 | 10.0 | 5.0* | 9–13 | 11 | 10 x 11 | 8–9 | 0 | 2–3 | 1.2 |
| 5.0 | 1.2 | 9 | 1.2 | 1.5 | 15.0 | 6.0* | 6–9 | 7 | 7 | 5 | 0 | 2 | 1.1 |
| 12.0 | 3.7 | 14 | 3.7 | 3.4 | 10.0 | 3.0* | 10–14 | 12 | 7 x 11 | 6–7 | 1 | 5–6 | 2.9 |
| 18.0 | 4.5 | 16 | 4.5 | 4.2 | 9.0 | 14.0* | 12–16 | 13 | 9 x 12 | 8–9 | 1 | 4–5 | 3.6 |
| 6.0 | 1.4 | 15 | 1.2 | 1.4 | 7.0 | 4.0* | 12–15 | 14 | 9 x 13 | 8 | 1 | 6 | 1.1 |
| 8.5 | 1.7 | 14 | 1.5 | 1.6 | 12.0 | 12.0 | 8–11 | 9 | 7 x 8 | 5 | 1 | 4 | 1.2 |
| 8.0 | 2.2 | 14 | 2.2 | 1.6 | 17.0 | 4.0* | 10–14 | 13 | 8 x 9 | 7 | 4 | 6 | 1.4 |
| 10.0 | 2.5 | 15 | 2.7 | 2.7 | 12.0 | 7.0* | 10–15 | 12 | 9 x 12 | 8–9 | 0 | 3–4 | 2.2 |
| 10.0 | 3.0 | 14 | 3.2 | 3.0 | 22.0 | 6.0* | 10–14 | 12 | 8 | 7 | 4 | 5 | 2.2 |
| 6.0 | 2.2 | 13 | 2.5 | 2.2 | 20.0 | 8.0* | 9–13 | 10 | 9 | 7 | 1 | 3 | 2.0 |
| 7.5 | 2.5 | 14 | 2.5 | 2.2 | 17.0 | 5.0* | 10–14 | 12 | 7 x 11 | 6–7 | 1 | 5–6 | 1.7 |
| 7.0 | 1.7 | 13 | 2.0 | 1.5 | 10.0 | 10.0 | 8–12 | 9 | 7 x 9 | 6–7 | 0 | 2–3 | 1.1 |
| 12.0 | 2.5 | 15 | 2.7 | 2.5 | 27.0 | 6.0* | 11–15 | 13 | 8 x 10 | 8 | 3 | 5 | 2.0 |
| 13.0 | 2.5 | 13 | 2.5 | 2.2 | 17.0 | 10.0* | 11–13 | 12 | 10 x 12 | 8–9 | 0 | 3–4 | 1.9 |
| 13.0 | 3.7 | 14 | 3.7 | 3.0 | 10.0 | 15.0* | 12–14 | 12 | 7 x 10 | 6–7 | 2 | 5–6 | 2.6 |

* Down to ground line.

the infection developed sufficiently on the stem to cause the annual accretion of wood to become abnormal.

By subtracting the greatest number of rings actually formed at the center of the canker from the theoretical age of the stem at this point, which, as explained in the third preceding paragraph, was derived from a count of the whorls of branches and checked by a ring count made on the normal wood, either above or below the canker, and corrected for the loss or gain of age incurred by making the count either one year's growth above or below respectively, there will be found in a number of cases a difference of as many as one, two, three, and even four years. No evidence of any false ring formation was detected in any of the transverse sections and, furthermore, the age of each stem at the point of its infection was checked by a count, made from the top down, of the whorls of branches. This difference between the theoretical and actual ages of the stem at the center of the canker can therefore be explained only on the ground that the growth of the wood around the stem had ceased in the vicinity of the center of infection, but that the increment continued on the stem both above and below the canker. It is likely that, after a certain length of time, the cambium about the canker was too badly weakened to make any further contribution to the xylem portion of the stem, or, if any further growth took place, it was so slight that its detection was impossible macroscopically. Normal additions to the stem, however, continued to be put on both above and below the canker. From this it is evident that there was no complete girdling of the stem at the center of the canker. The power of cell division of the cambium about the canker had practically or entirely ceased, unless we consider the possibility of the formation of a secondary phloem.

The above measurements indicate that the retention by the stem of its conductive power varies from one to as many as four years after the cessation of any material wood formation at the center of the canker before the death of the tree ensued. It is this cessation in the growth of the xylem at the center of the canker, and its continuance both above and below the lesion, that cause the marked constricted appearance commonly exhibited by infections of long standing on the older stems despite the partial compensation afforded by the presence of the greatly thickened bark covering the lesions.

Continuing with table 2, the column marked "Years required to kill tree after canker spread to main stem" is obtained by subtracting the figures in the column headed "Probable age of stem at center of canker at time of infection" from that headed "Theoretical age of stem at center of canker." For the reasons mentioned in the third preceding paragraph, this result probably is too low by at least two years. The results given, however, are approximate and indicate that the white pine blister rust, on the larger stems at least, is a comparatively slow-working disease, although nevertheless a very potent factor in killing young white pines.

The column headed "Average diameter of infected wood at center of canker" gives the diameter of the stem inside the bark on the transverse section made at the center of the canker, and, when subtracted from the column giving "Actual diameter" and the result divided by two, some idea may be had of the thickness of the bark on old blister rust cankers for stems of different sizes.

## BIOLOGIC RELATION OF COMPETITIVE SECONDARY FUNGI TO THE WHITE PINE BLISTER RUST

As was noted by Messrs. G. B. Posey and G. F. Gravatt during their work at Kittery Point in the summer of 1917, a number of miscellaneous secondary fungi frequently play an important rôle in interfering with the normal development of the blister rust on white pine. Not infrequently such secondary fungi may be so rapid in their growth that they overrun the blister rust lesion and completely suppress the rust so that it fails to develop pycnia and aecia. As a general rule the blister rust lesion invaded by the secondary fungus becomes constricted, and often very sharply so, even on small stems. In general it may be said that lesions caused by these so-called "secondary fungi," either alone or in combination with the blister rust, present quite a different appearance than lesions caused by the blister rust alone. From numerous observations made on the occurrence of these secondary fungi associated with the blister rust, it is evident that certain of them are quicker and more potent agencies in girdling the branch or stem than the blister rust alone. The majority of them, however, are very weak parasites and only retard the fruiting and rapidity of the mycelial growth.

## SCOLYTID ATTACKS RESPONSIBLE FOR HASTENING THE DEATH OF WHITE PINES INFECTED BY BLISTER RUST

As a contributing cause of the accelerated death of white pine trees infected by blister rust, the rôle of a scolytid beetle, *Pityogenes hopkinsi* Swaine,[2] is of considerable importance, at least in the vicinity of Kittery Point. On May 10 the writer first noted the presence of the adults on the trunks and branches of young white pines, and in a few days they were present in comparatively large numbers, beginning the work of excavating their tunnels, which resulted in the formation of numerous little heaps of "sawdust" all over many of the trunks. About three weeks after this the insect-infested trees suddenly commenced to die rapidly. Few other white pines on this area died at this time. Even those practically girdled by the blister rust, but not attacked by the scolytids, did not die as a rule until mid-summer, when the transpiration pull on them was greater, and then death was a comparatively slow and gradual process compared to that of the white pines attacked by insects. A careful study of the effect of *Pityogenes hopkinsi* in relation to the blister rust epidemic on the area under study showed that the attacks of this scolytid were limited entirely to such white pines as had their vitality more or less lowered by the presence of one or more blister rust lesions on the vital part, namely the trunk. In some cases the lesions had been invaded previously by a secondary fungus which had completely retarded the production of aecia. Such a relation between the general lack of vigor of the host tree and its attack by this species has been indicated by Blackman (1), although the white pine blister rust was not mentioned as one of the causes contributing to the insect attack. The attacked trees were all young ones, ranging in diameter from 1 to 4 inches and still retaining the thin smooth bark characteristic of young white pines. Many of the trees attacked by the beetles had not then been seriously affected by the blister rust. At the time of their attack by the insects, several still presented a thrifty appearance, and, without the interference of the insects, would not have died for at least from one to three years. *Pityogenes hopkinsi*, therefore, may be responsible at times for expediting the death of white pines infected by the blister rust.

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[2] Determination by Dr. A. D. Hopkins of the U. S. Bureau of Entomology, Washington, D. C.

## SUMMARY

From the behavior of three hundred aecia under study on various cankers it is evident that they attain the climax of their development within less than two weeks after the peridia start to open, and then begin to decline rapidly. Aeciospore dissemination may be regarded as practically at an end within four weeks after its start, the heaviest part being over within two weeks under normal conditions.

During the period from May 1 to September 10 the vertical growth of the blister rust cankers, both up and down, averaged 5 inches. During the same period the lateral growth, both to the left and right, averaged 2.5 inches, or one-half that of the vertical growth. It is likely that these figures represent about two-thirds of the total annual growth of the blister rust cankers.

Growth of the cankers during the latter part of the summer was twice as fast as that during the spring and early part of the summer. It is likely that the rate of growth of the blister rust on white pines is influenced chiefly by temperature.

The vertical growth of the blister rust cankers, when correlated with the size of the stems on which they occurred, indicates that the larger stems, up to 4 inches in diameter, the greater is the growth of the cankers. There is also shown for stems of 3 inches or less in diameter a general tendency for the downward growth to exceed the upward growth in cankers falling into the same diameter class.

A stem analysis of white pines killed by the blister rust reveals that infected twigs and smaller stems, up to about 2 inches in diameter, exhibit swelling which is proportionately greater on the smaller stems and solely due to the abnormal thickening of the bark. In the case of cankers of long standing on the older stems, usually those of 2 inches or more in diameter, however, the stem rarely becomes swollen but usually remains normal in outline or, in cankers of especially long standing, becomes constricted. The latter often exhibit a marked diminution in the increment of the stem at the center of the canker, which is often compensated for, in so far as the outline of the stem is concerned, by the abnormally thickened bark.

In the older cankers the number of growth rings formed at the center of the canker may be one, two, three, or even four, years less than the theoretical age of the stem at this point, based on the age of the wood both above and below the canker. This indicates that the retention by the stem of its conductive power may vary from none to as many as four years after the cessation of any material wood formation at the center of the canker before the death of the tree ensues.

A number of miscellaneous secondary fungi play an important rôle in interfering with the development of the blister rust. Such fungi, which are mostly of the nature of weak parasites, may be so rapid in their growth as to overrun the blister rust lesion and retard, more or less completely, the production of pycnia and aecia, as well as the growth of the mycelium.

Attacks by a scolytid beetle, *Pityogenes hopkinsi*, were responsible for expediting the death of a number of white pines infected by blister rust. The attacks of this beetle were limited entirely to such white pines as had their vitality more or less lowered by the presence of one or more blister rust lesions on the trunk.

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# A FOURTH PHYTOPHTHORA DISEASE OF TOMATO

DONALD REDDICK

Three diseases of tomato (*Lycopersicon esculentum*) caused by species of *Phytophthora* have been described. These are blight,[1] caused by *P. infestans*, buckeye rot of fruit[2] caused by *P. terrestria*, and foot rot or collar rot,[3] caused by *P. cryptogea*. A fourth disease caused by a species of *Phytophthora* occurs on tomatoes grown under glass in the vegetable houses at Cornell University. Plants of both winter and spring crops have been somewhat affected each year for a number of years. The disease has been noted by several persons but has not been an object of more than passing study until now.

*Name.* This disease has so many manifestations that a single name is not comprehensive enough to include all phases. The most characteristic symptom is a girdling of the stem and for convenience the name stem-girdle will be used in referring to it.

*Symptoms.* Briefly, the following symptoms may be noted: (a) typical damping off of seedlings; (b) a girdling of stems of all ages and at any point, with subsequent collapse of the plant above the lesion; (c) blight of foliage, usually in limited areas but occasionally involving one or more leaflets; (d) a rapid rot of fruit; (e) gradual death of foliage from the base and eventual death of the plant as a result of late root infection.

*Distribution and losses.* There seems to be no other record of the occurrence of this disease either in greenhouses or in fields. I. C. Jagger states, in correspondence, that the disease was not encountered by him in his rather extended investigations of truck crop diseases in New York, including observations made in the greenhouses at Irondequoit, New York, a region of intensive production under glass. In the houses at Ithaca the losses have been estimated at times as involving half the crop—enough to make commercial production unprofitable. It is likely, however, that other factors may be involved in this estimate, particularly the occurrence of leaf mold (*Cladosporium fulvum*) on the spring crop.

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[1] Reed, Howard, S. Tomato blight and rot in Virginia Virginia Agric. Exp. Sta. Bull. 192, 16 p 1911. See also, Giddings, N. J., and Anthony Berg. A comparison of the late blights of tomato and potato. Phytopath 9: 209–210, pl. 11. 1919

[2] Sherbakoff, C. D. Bu'ckeye rot of tomato fruit. Phytopath 7: 119–120, 5 fig. 1917.

[3] Pethybridge, George H , and H A. Lafferty. A disease of tomato and other plants caused by a new species of Phytophthora. Sci. Roy Dublin Soc. 15: 487–505, pl. 45–48. 1919.

*Cause.* The disease is caused by a fungous pathogene of the genus *Phytophthora.* That this is true is shown by detailed experimentation, results of which are recorded on subsequent pages.

The fungus has been studied in some detail and has been compared in culture and on the natural host with *P. terrestria* and *P. cryptogea*, cultures of which were kindly furnished by Doctors Sherbakoff and Pethybridge, respectively. It seems to the writer unquestionably distinct from these species and in general seems more nearly related to *terrestria* than to *cryptogea.* When it became known that Doctor Sherbakoff is engaged in a comparative study of several species of *Phytophthora* it seemed best to ask him to include consideration of the stem-girdle fungus. This he has consented to do so that all details regarding morphology, relationship, life history, and so forth will await this critical study. Certain details of special significance from the pathological standpoint are included here.

The peculiar swellings and distortions of the mycelium described for *P. cryptogea* are to be found regularly in cultures of the stem-girdle fungus in beef bouillon or beef-agar with sugar. Similarly, sporangia are produced very sparingly by this fungus, but if a fairly large bit of diseased tissue is transferred to a poured plate a few sporangia are very likely to develop on the tissue but not in the surrounding medium. Also if Clinton's formula[4] for making oat agar is followed a medium is secured on which appears a luxuriant growth of mycelium with an abundance of sporangia. Similar development of sporangia may be secured by breaking open an affected fruit and holding it in a moist chamber for a few days. Sporangia have not been observed in nature but if an infected stem is stood in a flask of water for a few days, a profusion of sporangia are produced near the surface of the water upon and about the lesion.

When mature sporangia are transferred to a drop of water, emission of swarm spores may be expected to begin within 10 minutes. This corresponds with the behavior of *P. terrestria* under similar circumstances. Swarming lasts less than an hour (at 20°) and germination of the swarm spore by a tube follows very promptly. Under favorable conditions the whole process, including the production of a tube long enough to reach through one cell of parenchyma in a tomato stem, might require so short an interval of time as from sixty to ninety minutes.

When cultures are a few weeks old, bodies develop on the mycelium which have the general appearance of the "clamps" of basidiomycete mycelium. These enlarge rather promptly, the protoplasm in the vicinity masses here, the wall becomes considerably thickened and the body takes on the general appearance of a chlamydospore with empty tubes issuing from two points which usually are not diametrically opposite. These bodies are in fact intercalary sporangia. They germinate by the production of swarm spores, the dehiscence being at one point of issuance of the original hypha. Germination occurs as promptly as in the case of sporangia produced exogenously but viability is retained longer, at least, in culture.

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[4] Connecticut Agric. Exp. Sta. Rept. **1909/10**: 760. 1910.

Oospores have not been found in nature but have developed sparingly on oat agar. Their development is not a factor in perpetuating the fungus in reference to greenhouse culture, since the mycelium itself remains alive for several weeks even on the limited amount of food contained in a tube of nutrient agar, and the intercalary sporangia may persist for several months.

*Isolation and growth.* The fungus is readily isolated from diseased tissues. In the majority of instances it can be obtained in pure culture simply by removing bits of diseased tissue, with aseptic precautions, to tubes or plates containing a suitable nutrient medium. Occasionally, bacteria appear with the fungus, but this is due to the fact that the dead tissue is rather rapidly invaded by bacteria and that tissue too long dead has been transferred. The fungus grows readily in a number of media: nutrient bouillon, the same with 1 per cent agar, oat agar, corn meal agar, bean pods, and the like. Better growth is made on soft agars than on hard ones. On corn meal agar a tufted growth similar to that made by *P. terrestria* appears but when the two are grown together it is found that the *Phytophthora* of stem-girdle grows more rapidly than *P. terrestria* and does not make such pronounced tufted growth. On beef agar with sugar, on the other hand, the thallus produced by the fungus resembles exactly Sherbakoff's illustration of *P. terrestria* when grown on corn meal agar.

*Inoculation and infection.* Preliminary trials of inoculating young plants, through wounds, in the manner described by Pethybridge and Lafferty, showed that the fungus would grow rapidly in succulent tissue and cause the breaking over and death of young plants in forty-eight hours. In order to test plants of various ages and to determine under what conditions infection might be expected to occur in nature, over 100 seedling plants of a forcing house strain of the variety Earliana were started in August, 1919. Plants were inoculated at intervals beginning September 11 and ending in February, 1920, the last plants inoculated being very large and with mature fruit.

As a result of the tests it is found that, other conditions being favorable, high humidity for a period not exceeding 15 hours (shorter periods not tested) assures infection. Every inoculation made in wounds on plants and protected by moist chamber resulted in infection. Some such inoculations, made on days of very high humidity, without the use of moist chamber were successful. Mycelium of the fungus either alone or on bits of agar was placed on uninjured surfaces of stem, leaf and fruit of all ages but infection did not occur under any conditions. Inoculations made with swarm spores, however, resulted in infection on leaf, tender stem and on fruit. The fungus was repeatedly reisolated from artificially produced lesions.

Usually, inoculations were made in two or three places on the stems—near the base, near the tip and sometimes at an intermediate point. Until the plants were 60 to 70 cm. tall the time required for the fungus to girdle the stem and allow it to break over was about the same. Even when the plants were a meter or more in height the difference in time was only such as could be accounted for by the greater distance the fungus must travel in order to girdle. All the plants were growing vigorously. Later, inoculations in plants which had become hard and stunted because left in small pots, and in the base of large plants growing in rich soil, showed that the fungus is materially retarded in its progress under such conditions although infection apparently occurred as readily as previously. Tips of large plants which were thick and succulent were girdled in characteristically short time. In all of the infection work, and in nature, it has been observed that the lesion almost invariably encircles the stem but that it does not extend vertically more than 10 or 15 cm.

In order to test the ability of the fungus to cause damping off, two plants which had become affected near the surface of the ground were cut off and seed scattered over the surface of the soil in the pots. Fresh soil was added to cover the seeds. Practically all the seeds germinated (30 or 40) but the seedlings gradually disappeared with characteristic symptoms of damping off and in a month a single plant remained. Subsequent test showed that it was not a resistant plant. The stem-girdle fungus was recovered from some of the seedlings that had damped off. A control pot showed no damping off of seedlings.

Plants of some size when transplanted to contaminated soil may persist for a long time. This is readily accounted for in that the fungus does not move vertically for a great distance in the tissue and especially in that the tomato plant pushes forth new roots in abundance and on slight occasion. But such plants rarely produce profitable crops of fruit.

Infection of fruit in all stages of development is secured easily by inoculating with mycelium in slight needle pricks or by suspending swarm spores in drops of water protected from evaporation. Infected fruits have not been observed in nature. Fruits in the same stage of development when inoculated with *P. cryptogea, P. terrestria* and the stem-girdle fungus are infected in about the same length of time but the latter passes through the fruit more rapidly. It does not produce the intense discoloration of the tissues caused by *P. terrestria* and the dark brown bands on the surface are not present. Instead the surface discolorations consist of blanched or light brown areas. Affected fruits fall off very easily but in one instance a fruit clung until it turned dark brown all over and the fungus passed back through the pedicel and caused a girdling of the stem.

Natural leaf infections have not been observed either but they might be overlooked very easily. Artificially infected leaves wither promptly but in practically every instance infection is limited to the inoculated leaflet.

*Inoculation on other hosts.* Pethybridge and Lafferty have produced disease in the following plants by inoculation with *P. cryptogea;* petunia, aster, wall-flower [*Cheiranthus*], *Gilia tricolor*, and *Fagus sylvatica;* also in potato tubers, apples, white turnips, mangels and swedes. The stem-girdle fungus possesses similar polyfagous tendencies. Characteristic pink-rot of potato tubers was secured, but inoculations on young plants (several trials at different times) were without result. Young plants of Navy pea bean were inoculated by making small slits in the stem near the base and inserting mycelium. Evident discoloration ensued and was present without appreciable enlargement until the plants matured seeds. Wounds in plants not inoculated calloused over without discoloration. Young pods of Navy pea bean were inoculated in slight injuries. In 24 hours watery lesions were evident and in 4 days the pods were nearly destroyed. Lesions did not extend back into the pedicel or stem and pods not inoculated eventually matured.

Leaves of squash plants (*Cucurbita pepo,* scallops) ready to set first fruit were cut off and mycelium of the stem-girdle fungus placed on the wounds. Surface cankers a few centimeters in length developed in 3 out of 4 inoculations and in the course of a few weeks the fungus was recovered in pure culture from the largest lesion.

The limited experience with squash, lead to some trials with cucumber[5] (*Cucumis sativus*), a plant which is commonly grown under glass and not uncommonly in rotation with tomato. These were not as extensive nor as satisfactory as could have been desired, probably because the plants were not really of a forcing variety. About a dozen inoculations were made through wounds and under fairly satisfactory conditions, although the plants were somewhat less than 20 cm. in length and already bore flowers.

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[5] A canker disease of cucumbers was observed years ago at Ithaca. See Atkinson, Geo. F. Canker in cucumber. *In* Cornell Univ. Agric. Exp. Sta. Bull. 94, p. 264. 1895. The organism was not definitely identified but it was thought to be *Pythium debaryanum.*

A few plants were practically girdled, a few showed no definite sign of infection but most of them developed a superficial lesion about the point of inoculation. These trials lead to the suspicion that the tomato fungus may persist in greenhouses on a crop grown in rotation with tomato.

Inoculations were made on 6 young eggplants (*Solanum melongena*), each with about 8 leaves, by removing 1 leaf and applying to the wound mycelium of the tomato fungus which had grown for six days in corn meal agar in a Petri dish. The plants were placed in a large moist chamber for 15 hours. In 8 days small lesions were apparent on all; and 1 plant showed signs of wilting. A week later the stem of this plant was girdled, the bundles were dark, the pith nearly black and the new leaves entirely dead and dry. The organism was recovered pure by a transfer of diseased tissue to a poured plate. The remaining five plants showed definite sunken lesions at the point of inoculation but they continued alive for several weeks and were finally discarded.

*Botrytis.* The description and illustrations of stem lesions in Stone's account[6] of a new tomato disease, on account of great similarity, might be reproduced here in connection with the *Phytophthora* disease. Considering the fact that various species of *Botrytis* have been used in the same and adjacent houses in which the infection work was performed it is very remarkable that *Botrytis* did not appear once in any phase of the work.

*Conditions affecting occurrence and spread of stem girdle.* The climatic conditions mentioned by Stone[6] for Toronto exist also at Ithaca In fact, Ithaca, New York, is known to the Weather Bureau[7] as one of the cloudiest places in eastern United States. The weak light coupled with artificial conditions under glass results in a tomato plant with broad leaves and thick succulent stems. The experiments just reported indicate that plants grown under such conditions are very susceptible. It is not clear, however, that these are not the ideal conditions for forcing tomatoes.

Coming to the greenhouse there are certain conditions and practices which favor spread of the disease. Tomato plants for the production of winter crops are started early in the autumn, usually some time before heat is required. In the same way the spring crop is being harvested at a time when heat is no longer needed. At these times and under the existing climatic conditions, humidity in the houses becomes high and remains so for hours or even days at a time. Such conditions assure infection once inoculation has been effected. It seems evident that the *Phytophthora* of stem-girdle does not form sporangia abundantly in nature for in spite of the occurrence of conditions just mentioned, natural infection of fruit and foliage has not been observed.

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[6] Stone, R. E. A new stem-rot and wilt of tomatoes. Phytopath. 9: 296–298, 2 fig. 1919.

[7] Oral communication by W. M. Wilson, District Forecaster for northeastern United States. See also an anonymous communication: Sun shines sometimes, its fair somewhere. Florist's Review, January 14, 1915, p. 9–12. The data apparently are compiled from Weather Bureau records but there is no indication whether they are for a single year or whether they are averages.

Natural infection on stems has been found to occur at points of injury. Such injuries may be accidental but most of them are an incident to the regular practice of disbudding or of removing waning leaves. Present indications are that mycelium from diseased tissue or contaminated soil is left on the wound from the knife or fingers of the workman.

Greenhouse conditions do not materially affect the occurrence of the disease on underground parts except that under such conditions the ordinary adversities of nature do not exist, so that it is likely the fungus persists there in greater amounts. The ability of the fungus to live on other hosts than the tomato also adds to the possibility of perpetuation.

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# RHIZOPUS INFECTION OF CORN ON THE GERMINATOR[1]

J. F. Adams and A. M. Russell

With Six Figures in the Text

The injury of storage organs of plants by *Rhizopus nigricans* under unfavorable conditions has been frequently reported. Seed infection with *Rhizopus nigricans* has been associated with certain symptoms but no evidence has been advanced as to the pathological histology. We have attempted to ascertain the manner in which this mould attacks the embryonic plant in the case of corn as a means of determining its degree of parasitism.

The testing of seed corn on germinators for selection of disease free seed corn has been an important factor for permanent corn improvement. During the past two years extensive germinating tests have been conducted at the Pennsylvania State College in charge of Professors C. R. Orton and E. L. Nixon. The germinating tests were run on a germinator similar in construction to that used by Hoffer and Holbert (10). During the progress of the work it has been observed that various types of moulds appeared to be a serious factor in the germination. One of the conspicuous and prevalent moulds under our conditions was *Rhizopus nigricans.* This fungus was very active under the favorable conditions of moisture on the germinator and appeared to be a serious agent in preventing the development of the seedling wherever it became established.

In recent years this fungus has been reported as the cause of rot in various fruits and vegetables. It was reported by Ehrenberg (3) as associated with the decaying fruits of mulberry, Cornelian cherry, and apple. The senior writer has observed it often with the decay of apples kept in unfavorable conditions of cellar storage. It has been reported causing the rot of tomatoes by Behrens (1), Wormold (18) and Hanzawa (6). Behrens produced infection with this fungus on pears, plums, raspberries and currants. Orton (13) and Hawkins (8) report it associated with a soft rot or "leak" of Irish potatoes. Halsted (5), Morse (11) and Edgerton (2) have reported it causing a rot of sweet potato and fruits. The fungus is now well known in this country as causing the soft rot and ringrot of sweet potatoes and leak of strawberries. Halsted (4) first associated it with the storage rot of sweet potatoes and in this connection it has received further investigation by Taubenhaus and Manns (17) and Harter, Weimer and Adams (7). Stevens and Wilcox (16) have conducted extensive investigations with the *Rhizopus* rot of strawberries and find that it causes a softening and rapid collapse of the strawberries. Stevens (15) finds that the mycelium is confined chiefly in the outer portion of the berry. "The hyphae grow between the cells, separating them and apparently extracting the cell sap. The nuclei of the cells persist unchanged until a late stage in the breaking down of the cytoplasm. When the fungus is grown on berries in a dry atmosphere, its action is somewhat different. The mycelium penetrates to the center of the berry, and hyphae are frequently found inside cells." Taubenhaus and Manns (17) illustrate the intercellular character and "vigorous mycelium" of *Rhizopus* from freshly infected sweet potato.

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[1] Contribution from the Department of Botany, Pennsylvania State College, No. 24.

FIG. 1. CROSS SECTION, SHOWING PORTION OF THE SCUTELLUM WITH THE PALISADE EPITHELIUM (*P*)

Below and between the "dark line" layers (*D*) the mycelium (*F*) is shown invading and separating this layer. At this point the mycelium is not shown penetrating this layer, while further to the right of this section it had penetrated. It is shown here (*F*) established above the "dark line" layer, and disorganizing the palisade of the epithelium (*P*). (× 500)

FIG. 2. CROSS SECTION OF THE SCUTELLUM SHOWING A POCKET OF MYCELIUM THAT HAS BECOME ESTABLISHED IN AN EPITHELIAL GLAND

The large irregular multinucleate mycelium is shown disorganizing the epithelial as well as the parenchymatous cells of the scutellum. (× 500)

FIG. 3. CROSS SECTION, SHOWING PARENCHYMATOUS CELLS OF THE SCUTELLUM JUST ABOVE THE PALISADE EPITHELIUM

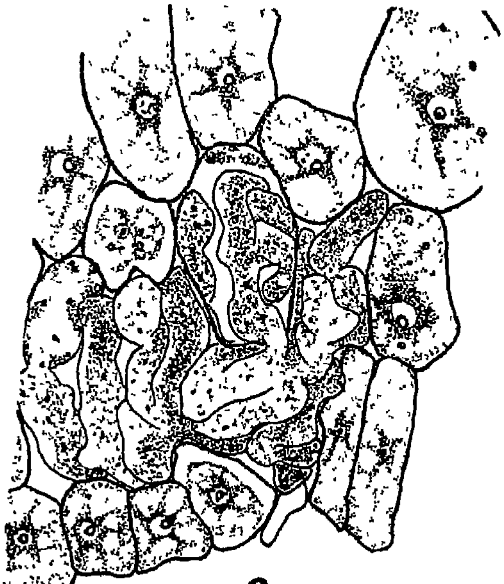
A pocket of mycelium has developed, disorganizing the tissue and one of the parenchymatous cells in the center is shown, penetrated by the mycelium. (× 600)

FIG. 4. PENETRATION OF PARENCHYMATOUS CELLS OF THE SCUTELLUM

The central host cell shows penetration by the hyphae. Plasmolysis has occurred and the cytoplasm (C) is seen adjacent to the hyphae. Other host cells also show plasmolysis of the cytoplasm. (× 500)

FIG. 5. PENETRATION OF THE PARENCHYMATOUS CELLS OF THE SCUTELLUM

The irregular multinucleate mycelium is shown in its method of invasion, with the characteristic constriction where it penetrates the cell wall. The contents of the cell on the left have disappeared. The cell to the right, which has been penetrated shows a remnant of the cytoplasm, pushed against the cell wall. (× 500)



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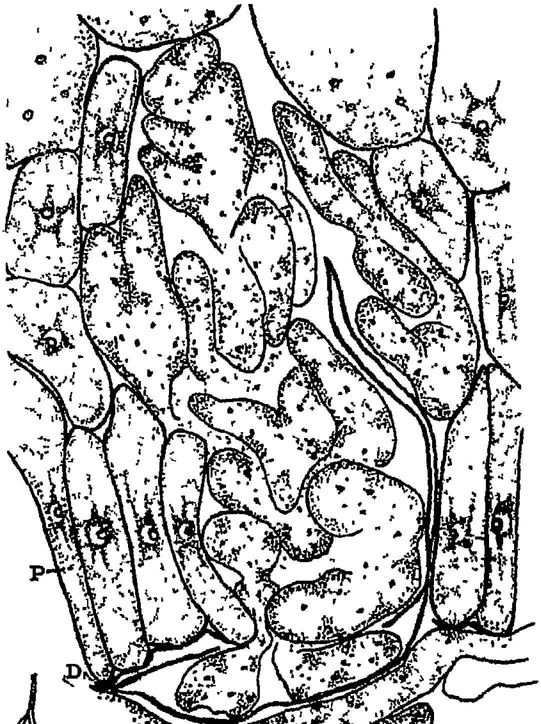

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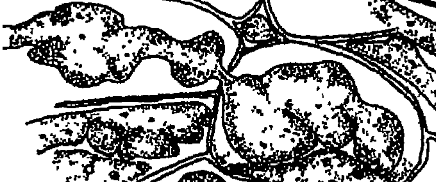

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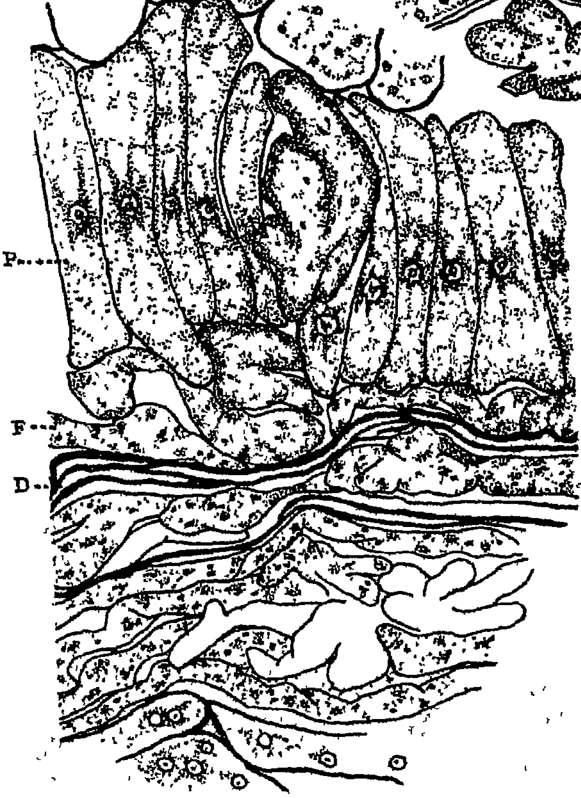

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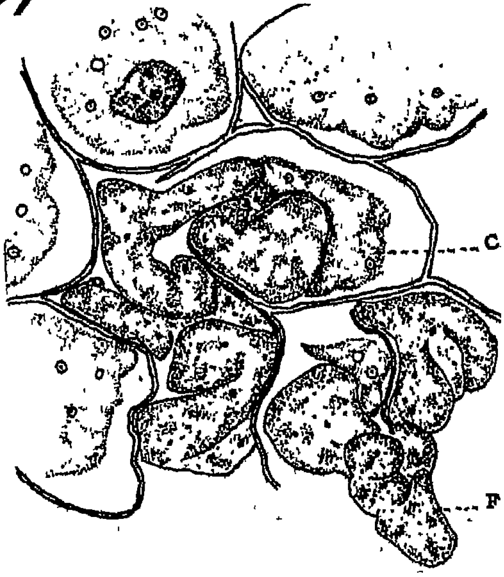

4

The importance of this mould in seed germination has been reported by Hiltner (9) and Muth (12). Hiltner observed in the germination of legume seeds, particularly lupine, that it was injurious to the sprout. In the fall of 1901 he found it especially injurious and active with the pot experiments in the greenhouse where the atmosphere was saturated with moisture. The young sprouts were killed off after the appearance of the mould. In the experiments conducted outdoors the mould was not active. Muth (12) found with his extensive germinating tests of various seeds that *Rhizopus nigricans* and *Cephalothecium roseum* were the common destructive moulds on the germinator. He states that *Cephalothecium roseum* was more prevalent and injurious than *Rhizopus nigricans*. Seeds of legumes and crucifers were more commonly infected than *Graminae* with the exception of *Zea mais*. *Rhizopus nigricans* and *Saccharomyces glutinis* were more injurious when inoculated together than separately.

The corn to be tested is placed between sterilized sheets of linen upon the germinator and watered from above. The appearance of the mould occurs about the time the radicle and plumule have broken the pericarp in the case of contaminated seed. From these foci it easily spreads under the conditions of moisture on the germinator and thus becomes established and involves adjacent seedlings. In two weeks time a large number of seedlings on the germinator may be affected.

With the rupture of the pericarp conditions become favorable for penetration of the embryo by *Rhizopus*. It is found with the kernel at this stage of development that the lateral margins of the scutellum become separable from the endosperm of the seed. The mould appears to first become established with the germinating seeds at this point, while the growth of the fungus is evident upon the surface. It is only with histological preparations that early infection is to be recognized. No internal symptoms are found on cutting embryos at this stage of development. Internal symptoms first become evident about the seventh day, when on cutting such seedlings a slight browning of the dorsal surface of the scutellum is seen. With further development of the embryonic plant a conspicuous blackening of the scutellum is found. This condition is evident by the tenth day. Along with the blackening of the scutellum further evidence of the infection is seen in the darkening of the vascular system of the cauline roots as well as the central stele. These internal symptoms are shown in text figure 6, which shows sections of infected kernels. These seeds were disinfected with corrosive sublimate (1:1000), placed in a moist chamber and inoculated with a sprayed suspension of spores of *Rhizopus nigricans*. They were removed and sectioned at the end of twelve days. These internal symptoms appear similar to Hoffer and Holberts (10) figures (15, 16, 17) illustrating corn rot although the exact cause of their symptoms is not mentioned. These authors however mention *Rhizopus* as one of the harmful organisms in connection with their corn root rot studies.

The result of infection upon the plumule is evidenced in the weakened condition and yellowed color of the shoots. Where the fungus grows up on the shoot a browning of the tissue and collapse of the shoot results.

For studying the pathological histology of the infected embryos material was fixed in Flemings or Petrunkewitsch killer and dehydrated in the usual way. Sections were cut 8 μ thick and stained with Fleming's triple by which the fungus is well differentiated from the host tissue with its characteristic multinucleated protoplasm.

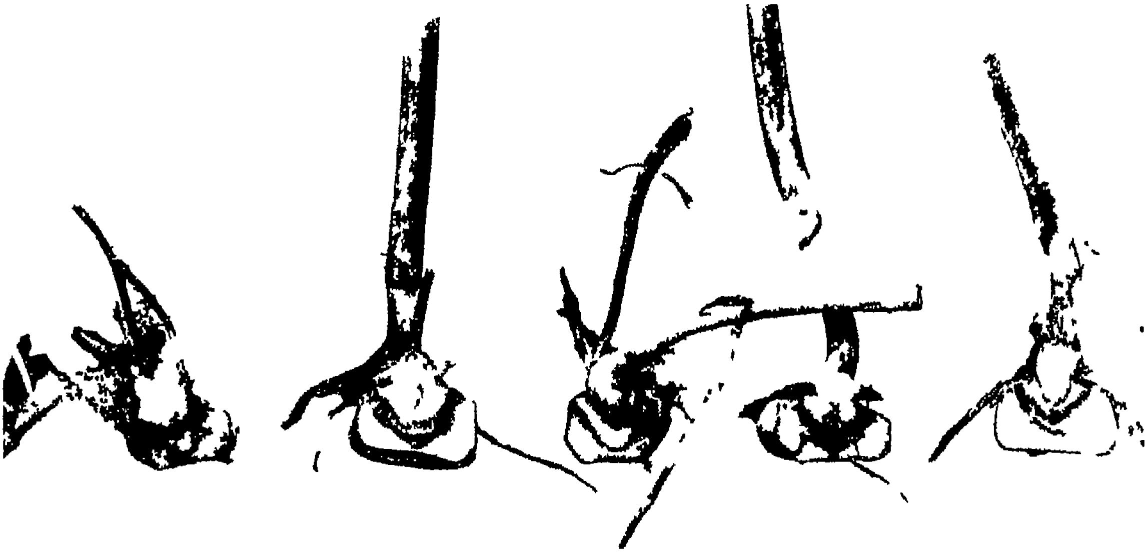

FIG 6. APPEARANCE OF INFECTED CORN KERNELS CUT AT EMBRYO END TO SHOW INTERNAL SYMPTOMS

A blackening of the scutellum as well as discoloration of the stele is evident. These kernels were disinfected and inoculated with a suspension of *Rhizopus* spores in a moist chamber. At the end of twelve days they were removed and sectioned to show the above symptoms.

As the fungus becomes established between the endosperm and dorsal epithelium of the scutellum it develops a thick interlaced mass of mycelium. This mass appears to act as a wedge in separating the embryo from the endosperm. The apparent mass action on the part of *Rhizopus nigricans* results eventually in completely separating the scutellum from the endosperm as cross sections through the embryo show a weft of mycelium completely covering the epithelium of the scutellum as shown in figure 6. The mycelium more distant from the epithelium becomes disorganized as only remnants of the mycelium void of protoplasmic contents are found.

Covering the dorsal epithelium is a dark layer often appearing in three strata. This layer appears to be similar to what Sargant and Robertson (14) refer to as the "dark line" appearance due to a viscous secretion from the epithelium cells. In some instances the mycelium acts as a wedge in separating this layer as shown in figure 6 or appears to penetrate it directly. After penetrating this layer the next resistance is found in the palisade layer of epithelium cells. The mycelium often pushes through between these cells into the inner tissues of the scutellum.

In the majority of cases the mycelium becomes established in the tissue of the scutellum by way of the glands. The glands are scattered over the whole surface of the epithelium except where it covers the top of the scutellum. These glands apparently allow for easy penetration of the tissue. Sections through the glands show them filled with the intertwined mycelium and they appear as "pockets of mycelium" as shown in figure 2. Extensive pockets may also be formed in cases where the hyphae have pushed between the epithelial cells at points where the glands are absent. From these points of penetration the mycelium usually ramifies as single strands. The parenchymatous cells of the scutellum are rich in stored food which appears to afford favorable conditions through which the fungus can ramify. In some instances the host cells are engulfed by the mycelium and show a conspicuous plasmolysis as the resultant effect of the fungus. The lateral vascular bundles are found surrounded by the hyphae as well as disorganized. All the cells of the epithelium appear plasmolysed where the mass of mycelium comes in contact with them.

Penetration of host cells by *Rhizopus nigricans* has been shown for disintegrated host cells of strawberry by Stevens (15) and of sweet potato by Taubenhaus and Manns (17). We have commonly found penetration of active host cells in the tissue of the scutellum. Various stages in penetration of host cells is shown in figures 3, 4 and 5. At the point of cell wall penetration the mycelium is constricted in some cases although in many instances it has penetrated the cell wall without any apparent constriction. The plasma membrane is not penetrated but pushed ahead of the hypha and in advanced stages the protoplast appears constricted and disorganized.

A typical necrotic symptom is associated with the growth of *Rhizopus nigricans* on the roots and plumule. These affected parts were fixed and sectioned but the preparations failed to disclose the hyphae penetrating the active growing tissue but the epidermal and some of the cortical cells were plasmolysed. In the case of the root cap the outer cells were found disorganized by the hyphae which had not deeply invaded this region. The abundant growth of the fungus on these parts is associated with a saturated atmosphere of moisture as found in the germinator or moist chamber.

The intercellular hyphae show considerable variation in size as they ramify through the tissues of the scutellum. The hyphae outside the host are much smaller. The intercellular mycelium appears more vigorous possibly because of its being in better relationship to the nourishment which is extracted from the host cells. This apparent enlargement may be caused by the mechanical resistance which prevents normal elongation resulting in the consequent thickening. The following measurements indicate the variations in width of hyphae outside and within the host tissue:

| | *Average* |
|---|---|
| Hyphae from agar plants | 9 μ |
| Hyphae in tissue external to scutellum | 10 μ |
| Hyphae within tissue of scutellum | 17 μ |

The scutellum is that part of the embryo directly infected by *Rhizopus nigricans*. During the germinating stage of the kernel the scutellum is important in nourishing the developing epicotyl and hypocotyl as well as relating them to the food supply of the endosperm. The infection of the scutellum prevents its normal functioning. The resultant effect is evident with the retarded growth, under-developed shoot and sub-normal color of the leaves. The infection by *Rhizopus nigricans* being restricted to the scutellum in the case of corn is further evidence that this fungus under favorable conditions may become parasitic upon parenchymatous tissue rich in food.

The sources of fungi and bacteria developing on the germinator has been discussed by Muth. His conclusions in this respect are indicated in part as follows: 1. Typical parasites established in or upon the seeds in the field. 2. Fungi and bacteria brought in upon seed and harmless in the field but becoming parasitic under conditions unfavorable to the seed such as occurs on the germinator. 3. Moulds from the air such as Rhizopus, Penicillium, Mucor, etc. 4. Fungi and bacteria naturally in water and those in sand and on apparatus.

It has been observed that fungi and bacteria present though harmless in the field will appear destructive on the germinator and thus be a factor in determining normality of germination. Eliminating the possibility for development of such species of fungi will give a better index of seed viability and produce better evidence of infection within the seed. In the case of *Rhizopus nigricans* this fungus is so ubiquitous that there are numerous sources for contamination of seed corn from the time the corn is harvested until it is tested upon the germinator. We have found that this contamination as well as that of various other fungi appearing on the germinator may be eliminated by seed disinfection and thus give a better index to normality of germination. Also by this method the presence of infection within the seed is now accurately determined. In our tests we have secured satisfactory results by soaking corn for three minutes in a 1:1000 solution of corrosive sublimate. By this means we have been able to hold in check the development of any contaminating organisms on the seed in the germinator as well as moist chamber. Where any fungus growth appeared or was associated with lack of germination the evidence indicated the presence of infection within the seed. A similar method for determining the presence of infection within the seed of legumes was used by Hiltner (9). This investigator sterilized seed for ten to twenty minutes in a 1 to 2 per cent solution of corrosive sublimate and then in absolute alcohol which was burned off. Infection within the seed was then determined by planting parts upon gelatin.

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# PHYTOPATHOLOGICAL NOTES

*The Crop Protection Institute.* On June 23 there was held at Rochester, New York, a meeting at which thirty-two delegates from the manufacturers of fungicides and insecticides, the National Research Council, the American Association of Economic Entomologists, and the American Phytopathological Society were present for the purpose of considering the feasibility of forming an association or organization of these respective interests to promote coöperative efforts. The meeting was called to order by Dr. P. J. Parrott. Dr. H. E. Howe of the National Research Council was elected chairman and Dr. G. R. Lyman, secretary. Definite action was taken by nominating an organization committee representing the four interests present. This committee drew up tentative articles of organization for a Crop Protection Institute, which were presented before the delegates present and adopted with certain recommended changes. The meeting was adjourned subject to the call of the chairman, after it was voted to call a meeting later for the purpose of adopting articles of organization.

On October 28 a meeting was called by Chairman G. R. Cushman and held in the offices of the National Research Council, Washington, D. C., Dr. H. E. Howe of the National Research Council presiding. At this meeting questions of purpose and policy were discussed and the former committee met and revised the tentative articles of association. These were then presented in open meeting and separately and collectively adopted.

The administration of the Crop Protection Institute is placed in the hands of a Board of Trustees consisting of thirteen members, three to be elected by the American Association of Economic Entomologists, three by the American Phytopathological Society, two by the Association of Official Agricultural Chemists, one by the National Research Council, and four by the manufacturers of fungicides and insecticides, machinery, and allied lines of industry. A director may be employed at the discretion of the Board of Trustees.

The constitution provides for four classes of membership: (1) Honorary, (2) Industrial, (3) Associate Industrial, and (4) Scientific. To the membership dues the industrial members pay $100 annually, associated industrial $10.00, and scientific members $1.00. Honorary membership is gratis.

The purpose of the Institute is:

(1) "To promote the general welfare through the efficient control of injurious insects and plant diseases affecting all economic and ornamental plants and their products."

(2) "To promote efficient control of insects and plants injurious to man, animals, and animal products."

(3) "To support and direct research upon other problems of similar nature."

(4) "To further coöperation between entomologists, scientific workers, plant pathologists, and the manufacturers of insecticides, fungicides, and similar materials, and the manufacturers of appliances required for their use."

(5) "To assist in the dissemination of scientifically correct information regarding the control of injurious insects and plant diseases."

"Any funds required for the pursuit of specific scientific or educational projects or for other purposes shall be secured, received, and allotted by the Board of Trustees. No assessment shall be levied upon the industrial members except upon approval of the Board of Trustees and a majority of the industrial members of the Institute."

"Any member of the Institute shall have the right to suggest as many problems as may be of interest, and with these suggestions before them the Executive Board will determine the scientific work to be carried out at the expense of the Institute. The results of such work shall be made available to all members of the Institute and generally published."

"If the problems suggested cannot be recommended by the Board of Trustees for investigation at the expense of the Institute, the member or group of members presenting the subject for research may still arrange with the Board at its discretion to have the undertaking carried on at his or their cost, this cost to be determined by the Board of Trustees. Publication of results are to be at discretion of Board."

"The published work of the Institute may be placed either in existing journals or in a special publication as may later be determined by the Institute."

Temporary appointments to the Board of Trustees were made pending regular election by the separate organizations represented in the membership. These elections will be effected by the scientific membership at their next annual meeting. Correspondence concerning the Crop Protection Institute should be addressed to Dr. H. E. Howe, Secretary, National Research Council, Washington, D. C., or Prof. W. C. O'Kane, Temporary Chairman, Durham, N. H.

C. R. ORTON

*Personals.* Dr. W. B. Brierley, Mycologist, Rothamsted Experimental Station, Harpenden, England, and Professor Etienne Foex, Director of the Station de Pathologie Végétale, Paris, France, accepted the invitation of the American Phytopathological Society to attend its field conference on fruit diseases, held in the Shenandoah Valley of Virginia and West Virginia and the Cumberland Valley of Maryland and Pennsylvania, August 3 to 6.

Following the field conference, both Doctor Brierley and Professor Foëx visited a large number of institutions in the United States, conferring with plant pathologists regarding the pathological investigations in this country. Professor Foëx sailed for France September 8. Doctor Brierley sailed for England October 9.

Dr. C. D. Sherbakoff has accepted the position of pathologist at the Tennessee Agricultural Experiment Station.

The following officers of the Pacific Division of the American Phytopathological Society have been elected and will hold office for two years: *President*, Dr. H. S. Reed, Riverside, California; *Vice president*, Dr. J. W. Hotson, University of Washington, Seattle; *Secretary-Treasurer*, Dr. S. M. Zeller, Oregon Agricultural College, Corvallis, Oregon.

# LITERATURE ON PLANT DISEASES[1]

COMPILED BY ALICE C. ATWOOD AND FLORENCE P. SMITH, BIBLIOGRAPHICAL ASSISTANTS, BUREAU OF PLANT INDUSTRY

## September, 1920

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[1] This list includes references, both American and foreign, to the literature of plant pathology and mycology of interest to pathologists. Foreign references published since January 1, 1917, have been included beginning with the list appearing in v. 7, no. 3, June, 1917.

All authors are urged to cooperate in making the list complete by sending their separates and by making corrections and additions, and especially by calling attention to meritorious articles outside of regular journals. Reprints or correspondence should be addressed to Miss E. R. Oberly, Librarian, Bureau of Plant Industry, U. S. Dept. Agric., Washington, D. C.

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